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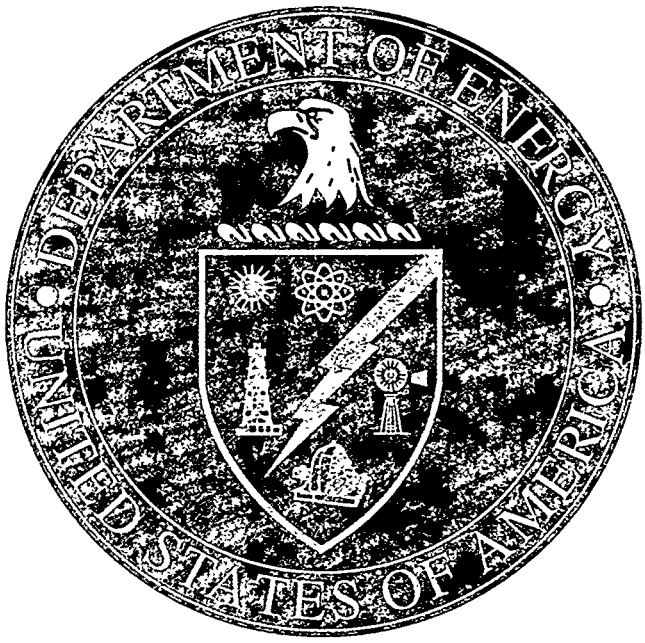
East Fork Poplar Creek – Sewer Line Beltway Remedial Investigation Report

Sections 5 - 8



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East Fork Poplar Creek – Sewer Line Beltway Remedial Investigation Report

Volume II
Sections 5-8

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ACRONYMS

This master list contains acronyms for all sections.
Not all acronyms on this list will appear in this volume.

AEA	Atomic Energy Act
AET	adverse effects threshold
AF	Adherence Factor
AFDW	ash-free dry weight
ALARA	as low as reasonably achievable
ANOVA	analysis of variance
AMS	Aerial Measuring System
ARAP	Aquatic Resources Alteration Permitting
ARAR	applicable or relevant and appropriate requirement
ASME	American Society of Mechanical Engineers
AT	Averaging Time
AWQC	ambient water quality criterion
B*a*	Biotransfer Factor from Feed to Beef Tissue
BAF	bioaccumulation factor
BC	Bear Creek
BCF	Bioconcentration Factor
BEIA	Biomedical and Environmental Information Analysis
BLS	Below Land Surface
BLUE	best linear unbiased estimator
B*m*	Biotransfer Factor from Feed to Milk
BMAP	Biological Monitoring and Abatement Program
BN	Brown-Norway
BRA	Baseline Risk Assessment
BTF	Biotransfer Factor
B*v*	Biotransfer Factor from Soil to Plant
BW	body weight
CAA	Clean Air Act
CAD	computer aided design
CAVEAT	Computer Assisted Validation and Evaluation Assessment Tool
CCC	criterion continuous concentration
CCS	Contract Compliance Screening
CDC	Centers for Disease Control
CDF	Cumulative Density Function
CEQ	Council on Environmental Quality
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFR	*Code of Federal Regulations*
CLP	Contract Laboratory Program
CMC	criterion maximum concentration
CMS	corrective measures study
COCs	contaminants of concern

COE	U.S. Corps of Engineers
Colex	column exchange
COR	Contracting Officer's Representative
CR	Chestnut Ridge
CRAC	Central Risk Assessment Council
CRAVE	Carcinogen Risk Assessment Verification Endeavor
CRDL	contract required detection limit
CSFs	Cancer Slope Factors
CVAA	Cold Vapor Atomic Absorption
CWA	Clean Water Act
CY	calendar year
D&D	decontamination and decommissioning
DCGs	derived concentrations guides
DER	duplicate error ratio
DO	dissolved oxygen
DOE	Department of Energy
DOE-OR	U.S. Department of Energy Oak Ridge Operations
DOI	U.S. Department of Interior
DOT	U.S. Department of Transportation
DQO	Data Quality Objective
EA	environmental assessment
EDE	effective dose equivalent
EFH	Exposure Factors Handbook
EFK	East Fork kilometer
EFPC	East Fork Poplar Creek
EG&G/EM	EG&G Energy Measurements, Inc.
EIS	environmental impact statement
EMSL	Environmental Monitoring Systems Laboratory
EO	Executive Order
EP	equilibrium partitioning
EPA	U.S. Environmental Protection Agency
ERA	ecological risk assessment
ERP	Environmental Restoration Program
FDA	U.S. Food and Drug Administration
FFA	Federal Facility Agreement
FFCA	Federal Facility Compliance Agreement
FR	*Federal Register*
FS	feasibility study
FWS	Fish and Wildlife Service
GIS	geographical information system
GN	Glomerulonephritis
HAs	Health Advisories
HAZWRAP	Hazardous Waste Remedial Actions Program
HC	Hinds Creek
HEAST	Health Effects Assessment Summary Table
HEC	Human Equivalent Concentration

HI	Hazard Index
HQ	Hazard Quotient
HSWA	Hazardous and Solid Waste Amendments
IAP	ion activity product
ICP	inductively coupled plasma
ICRP	International Commission on Radiological Protection
INM	in need of management
IRIS	Integrated Risk Information System
LCS	laboratory control samples
LDR	land disposal restriction
LEFPC	Lower East Fork Poplar Creek
Li	lithium
LLW	low-level waste
LOAEL	Lowest-Observable-Adverse-Effect Level
M&O	management and operating
MCL	maximum contaminant level
MCLG	maximum contaminant level goal
MF	Modifying Factor
MFO	mixed function oxidase
MLE	Most Likely Exposure
MOU	memorandum of understanding
MS	matrix spike
MSL	mean sea level
MSS	matrix spike sample
NAs	Natural Areas
NAS	National Academy of Sciences
NAA	neutron activation analysis
NCP	National Oil and Hazardous Substances Pollution Contingency Plan
NCR	Nonconformance Report
NCRP	National Council on Radiation and Protection Measurements
NEPA	National Environmental Policy Act
NERP	National Environmental Research Park
NESHAPs	National Emission Standards for Hazardous Air Pollutants
NHP	New Hope Pond
NHPA	National Historic Preservation Act
NMFS	National Marine Fisheries Service
NOAA	National Oceanic and Atmospheric Administration
NOAEL	No-Observable-Adverse-Effect Level
NPDES	National Pollution Discharge Elimination System
NPL	National Priorities List
NRC	U.S. Nuclear Regulatory Commission
NRDAs	Natural Resource Damage Assessments
NSDWS	National Secondary Drinking Water Standards
NSPSs	new source performance standards
NWP	nationwide permit
ODW	Office of Drinking Water

ORAU	Oak Ridge Associated Universities
ORGDP	Oak Ridge Gaseous Diffusion Plant
ORNL	Oak Ridge National Laboratory
ORR	Oak Ridge Reservation
ORSA	Oak Ridge Sportsman Association
ORSTP	Oak Ridge Sewage Treatment Plant
ORTF	Oak Ridge Task Force
OSHA	Occupational Safety and Health Administration
OU	operable unit
PA/SI	preliminary assessment/site investigation
PAHs	polycyclic aromatic hydrocarbons
PARCC	precision, accuracy, representativeness, completeness, and comparability
PCBs	polychlorinated biphenyls
PCDFs	polychlorinated dibenzofurans
PDF	Probability Density Function
ppm	parts per million
PRGs	preliminary remediation goals
QA	quality assurance
QAPjPs	Quality Assurance Project Plans
QAPP	Quality Assurance Program Plan
QAMS	Quality Assurance Management Staff
QC	quality control
RAs	Reference Areas
RAGS	Risk Assessment Guidance for Superfund
RCRA	Resource Conservation and Recovery Act
RfCs	reference concentrations
RfDs	reference doses
RFI	RCRA facility investigation
RI	remedial investigation
RI/FS	Remedial Investigation/Feasibility Study
RME	Reasonable Maximum Exposure
RMPE	Reduction of Mercury in Plant Effluents
ROD	record of decision
ROW	right of way
RPD	Relative Percent Difference
SAIC	Science Applications International Corporation
SAP	sampling and analysis plan
SARA	Superfund Amendments and Reauthorization Act
SCS	Soil Conservation Service
SDWA	Safe Drinking Water Act
SE	standard error
SEN	Secretary of Energy Notice
SI	saturation index
SLB	Sewer Line Beltway
SMCLs	secondary maximum contaminant levels
SO_2	sulphur dioxide

SOP	standard operating procedure
SQCs	sediment quality criteria
SVOC	semivolatile organic compound
SWMUs	solid waste management units
TBC	to be considered
TCA	Tennessee Code Annotated
TCL	target compound list
TCLP	toxity characteristic leaching procedure
TDEC	Tennessee Department of Environment and Conservation
TEF	Toxicity Equivalence Factor
THQ	Target Hazard Quotient
TIC	Tentatively Identified Compound
TIR	tentatively identified radionuclides
TOC	total organic carbon
TRL	target radionuclide list
TSC	Technical Support Contractor
TSCA	Toxic Substances Control Act
TSD	treatment, storage, and disposal
TSPs	total suspended particles
TU	trap units
TVA	Tennessee Valley Authority
TWA	Time Weighted Average
TWRA	Tennessee Wildlife Resources Agency
UCL	Upper-bound Confidence Limit
UEFPC	Upper East Fork Poplar Creek
UF	Uncertainty Factor
USGS	U.S. Geological Survey
URF	Unit Risk Factor
VOCs	volatile organic compounds
WQC	water quality criteria

5. BASELINE HUMAN HEALTH RISK ASSESSMENT

Risk assessment is an essential component of the remedial investigation/feasibility study (RI/FS) process at hazardous waste sites. The Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) and the National Oil and Hazardous Substances Pollution Contingency Plan (NCP)(EPA 1990a) require that actions selected to remedy hazardous waste sites be protective of human health and the environment. An overview of risk assessment in the RI/FS process is presented in the NCP and in the U.S. Environmental Protection Agency (EPA) manual *Guidance for Conducting Remedial Investigations and Feasibility Studies Under CERCLA* (EPA 1988a). A baseline human health risk assessment (BRA) is conducted as part of the RI to assess site conditions in the absence of remedial actions. As part of the FS process, risk assessment is used to evaluate the acceptability of proposed remedial actions and as a tool in developing remediation objectives (target cleanup levels).

A BRA has been conducted for East Fork Poplar Creek (EFPC) and the Sewer Line Beltway (SLB) in support of the RI process. The primary objectives of this assessment are to determine if there is an "imminent and substantial" endangerment to human health based on current and future exposure potential and to evaluate the need for site remediation. The risk assessment will examine the presence of chemicals in EFPC attributable to release from the U.S. Department of Energy (DOE) Y-12 Plant, the potential routes of exposure to human receptors, and the likelihood of adverse health effects following contact with contaminated environmental media.

A phased (iterative) approach to risk assessment has been adopted in conducting the BRA for EFPC. As noted, the overall goal of the BRA is to evaluate the risks to human health in the absence of site remediation. In accomplishing this goal, a hierarchical approach to risk assessment has been adopted that facilitates derivation of the most scientifically valid estimates of the potential for adverse effects. The primary objective has been to focus the evaluation on the receptors and exposure pathways of principal concern and to quantitatively characterize the uncertainty surrounding all assumptions and resultant risk estimates. The EFPC risk assessment team has worked with EPA Region IV, DOE, and the Oak Ridge National Laboratory Central Risk Assessment Council (ORNL CRAC) throughout the process in an effort to establish a consensus and ensure consistency with existing guidance. Recent EPA guidance for human health risk assessment includes *Risk Assessment Guidance for Superfund (RAGS): Human Health Evaluation Manual Part A and Part B* (EPA 1989a and EPA 1991c, respectively); *Human Health Evaluation Manual, Supplemental Guidance: Standard Default Exposure Factors* (EPA 1991a); *Supplemental Guidance to RAGS: Calculating the Concentration Term* (EPA 1992c); *Guidance*

for Exposure Assessment (EPA 1992d); *Health Effects Assessment Summary Tables (HEAST), Annual Update* (EPA 1992e); and *New Interim Region IV Guidance* (EPA 1992a).

The objective of this study has been to develop estimates of risk and the associated uncertainty that meaningfully project the potential for adverse effects in exposed individuals. Risk assessment has been conducted in three tiers:

- Tier 1: screening-level deterministic assessment using monitoring data from locations of (projected) highest concentrations,
- Tier 2: deterministic assessment using the full (creek-wide) data set, and
- Tier 3: probabilistic assessment and quantitative uncertainty analysis using the full data set and focusing on pathways that drive the overall risk assessment.

The quantitative uncertainty analysis will be conducted using Monte Carlo analysis and computer simulation software. These methods are presented in more detail in Sects. 5.2 and 5.5.

The human health risk assessment process, as outlined by EPA, is divided into four fundamental component analyses: data collection and evaluation, exposure assessment, toxicity or hazard assessment, and risk characterization. Although an analysis of uncertainty is conducted throughout the risk assessment, uncertainty analysis is often considered and presented as a fifth component of the risk assessment process. These fundamental elements form the basis of the BRA for EFPC and are presented in the following sections.

5.1 DATA COLLECTION AND EVALUATION

One of the first steps in the risk assessment process is to obtain and evaluate all available data on contaminants present in the environmental media in the EFPC system. This includes information on levels of chemical contaminants and radionuclides in soil, sediments, surface water, groundwater, and animal and plant tissue. Environmental monitoring data used in the baseline human health risk assessment have been obtained primarily from two sources: the Phase Ia sampling program and the Phase Ib sampling program. Section 3.2 summarizes the existing data for all environmental media and provides an overview of the sampling and analysis program. An additional garden study was conducted to determine chemical uptake by various plant species and to support the analysis of food chain pathways [see *Phase Ib - Sampling and Analysis Plan Addendum for Garden Produce and Vegetation* (Radian 1992b)]. The relevance and importance of this study are discussed in Sect. 5.2.

This section of the BRA focuses on the following elements:

- overview of the sample collection program and data quality assessment as it relates to the human health risk assessment,
- summary of methods and selection of contaminants of concern (COCs) for the Tier II risk assessment,
- derivation of exposure point concentrations, and
- discussion of uncertainty associated with the environmental monitoring data.

As discussed in the introduction, the human health risk assessment has been conducted iteratively in three stages, or tiers. The first tier makes use of the data set obtained from the Phase Ia sampling program. Based upon the results of both the Tier I analysis and the concentration-toxicity screen presented in Sect. 5.1.2, COCs were selected as the focus of the Tier II and Tier III risk analyses.

5.1.1 Review of Sampling and Analysis Program

The design of the sampling and analysis program was based on an understanding of the physical setting of the Y-12 Plant and the nature of contaminant release, as well as the transport and deposition of contamination within the floodplain boundaries. An initial understanding of the nature and extent of contamination in the EFPC drainage system was obtained from historical studies and is founded on a watershed-based approach to system dynamics (i.e., examination of the entire EFPC floodplain and all influents, excluding the Bear Creek drainage). The EFPC watershed unit includes areas above and below the Y-12 Plant extending downstream to the confluence with Poplar Creek and is defined to include the following media: air, land (surface and subsurface components), groundwater, and surface water/sediment.

The sampling program was designed to examine all areas potentially affected by releases from the Y-12 Plant. The most important releases of contaminants to the EFPC watershed are associated with the activities occurring within the confines of the Y-12 Plant. Overall, three sources of materials (deduced from historical studies) are responsible for contamination within the EFPC watershed: waterborne Y-12 migration from upper EFPC, non-Y-12 migration to the EFPC system, and outputs from the Bear Creek subsystem to EFPC.

No groundwater inputs to the EFPC watershed have been identified. Existing studies conducted on the Oak Ridge Reservation (Moore 1988) indicate that groundwater discharges to nearby streams and does not cross surface water divides. Therefore, the groundwater survey in the RI was limited to the EFPC watershed.

Sampling strategies for the EFPC floodplain system were devised with the following four objectives in mind:

- evaluate the movement (sources) of water, sediments, and contaminants into the EFPC watershed;
- examine the transport of contaminants within and between environmental media;
- evaluate the potential exposure of human and ecological receptors to contaminated media in the EFPC floodplain system; and
- examine outputs of water, sediments, and contaminants from the EFPC system.

The sampling and analysis program was designed to provide the highest quality data (equivalent to EPA's Level IV data quality) and to ensure that the data were technically and legally defensible. Field activities were conducted according to technical procedures approved under the Environmental Restoration Program (see Sect. 3.2 for a complete list) and all samples were tracked with full chain-of-custody and field documentation. QC samples (e.g., field duplicates and trip blanks) were obtained at periodic intervals established in the *East Fork Poplar Creek - Sewer Line Beltway Sample and Analysis Plan for Phase Ia* (LWA 1991). Field surveillance was conducted throughout the sampling activities to ensure adherence to protocol and procedures.

An analysis of contaminants in air has been conducted in this RI (Sects. 3.2.6 and 3.2.7), but not as part of the Phase Ia or Ib programs. In general, these analyses conclude that contaminants in air do not measurably impact the regional air quality. Therefore, exposure to this medium has not been evaluated in the risk assessment.

5.1.1.1 Sample collection

The sampling program for EFPC consisted of two phases or stages. The Phase Ia program consisted of an initial screening for 182 inorganic, organic, and radionuclide contaminants at three locations across EFPC. Samples were collected from surface water, sediments, soil, and groundwater. The second sampling program, Phase Ib, was a creek-wide assessment and evaluated contamination along the length of the entire EFPC system. Only selected contaminants were included in the analyses of the Phase Ib samples. Both the Phase Ia and Phase Ib programs are discussed in detail in Sect. 3.2.

For the Phase Ia program, three areas with the highest concentrations of mercury were selected to be the focus of the sampling: depositional wetland areas behind the National Oceanic and Atmospheric Administration (NOAA) Atmospheric Diffusion Laboratory off Illinois Avenue, across the Oak Ridge Turnpike from the Bruner's Shopping Center on the Wayne Clark property,

and the Mel Sturm property. The NOAA and Bruner's areas are inundated several times a year during high rainfall events.

Groundwater sampling and analysis were conducted as part of the Phase Ia program to examine the potential impacts (spatial and temporal) downstream and downgradient from the main source of release. The groundwater sampling program also was designed to evaluate potential effects in proximity to other attributes of the watershed (i.e., tributaries, urban features, and contaminant hot spots). Monitoring wells were sampled on four separate occasions to identify seasonal variations and contaminant migration patterns.

Surface water and sediment samples were collected at a number of locations: the point of input to the EFPC system (i.e., Lake Reality and the major tributaries within the watershed); the primary output location (i.e., the confluence with Poplar Creek); and intermediary positions within the system during both Phases Ia and Ib.

The Phase Ib investigation is described in detail in *East Fork Poplar Creek - Sewer Line Beltway Phase Ib Sampling and Analysis Plan for Soil, Sediment, and Water* (Radian 1992a). The Phase Ib program used the same analytical procedures and methods as Phase Ia for surface water, sediments, and select soil samples (groundwater was sampled only under the Phase Ia investigation, even though later rounds of sampling were conducted concurrently with Phase Ib). Soil samples collected during the Phase Ib sampling program also were analyzed for select metals and radionuclides using Neutron Activation Analysis (NAA) (Sect. 5.1.3.1 and Appendix I).

5.1.1.2 Sample analyses and validation

Samples were analyzed by a commercial laboratory participating in the EPA Contract Laboratory Program (CLP). Samples collected from the EFPC floodplain were analyzed for metals, radionuclides, volatile and semivolatile organic compounds (VOCs and SVOCs), and pesticides and polychlorinated biphenyls (PCBs) in soils, sediments, groundwater, and surface water.

NAA was selected as the primary method for analyzing metals and radionuclides in floodplain soils. NAA fully meets the data quality objectives (DQOs) of the investigation and is ideal for processing the large number of samples within a reasonable time and at a reasonable cost. Appendix I presents a report (*Comparison of Neutron Activation and Cold Vapor Atomic Absorption for the Analysis of Mercury in Soils from East Fork Poplar Creek*) detailing the equivalency of NAA to standard CLP methods. NAA-specific procedures and methods, including quality control (QC) measures, are outlined in the ORNL Analytical Chemistry Division Standard

Analytical Method AC-MM-222002, Neutron Activation Analysis of East Fork Poplar Creek Samples (copy attached to equivalency report in Appendix I). Results of the NAA were reviewed according to the Laboratory Data Validation Guidelines for Evaluating Neutron Activation Analyses (SAIC 1992a).

A percentage of the soil samples collected during Phase Ib were analyzed using standard CLP methods. CLP methods were used to evaluate levels of inorganic and organic chemicals in soils that are not measurable using NAA. Metals concentrations also were measured to provide a basis of comparison of concentrations determined by NAA. Although an exact agreement of concentrations between methods was not expected given the large degree of soil heterogeneity, a comparison of the concentrations showed similar levels of specific analytes and indicated additional confidence of equivalency between results.

Preliminary and final analytical results were incorporated into a data management system to facilitate the analysis, reporting, storage, and archiving of the data. The TP-DM-300 series of procedures for the Environmental Restoration Program provides guidance and protocols for data entry, data archival, data backup and recovery, sample identification, and data receiving and reviewing. Analytical results were screened under the technical procedure, TP-DM-300-7 *Data Package Verification and Validation*, to ensure that the resulting data would satisfy the highest level data criteria so that results could be incorporated into the BRA. Data that did not meet the criteria were qualified as usable or rejected (i.e., unusable). Data that were rejected were not incorporated into the BRA. Section 3.3 summarizes the results of the data validation and provides an assessment of the quality and completeness of the data used in this RI.

5.1.2 Selection of Contaminants of Concern

From the full list of all chemicals identified in the environmental media (Phases Ia and Ib), a subset has been identified for use in the risk assessment. In general, it may be impractical to evaluate all chemicals observed in environmental samples (due to limitations in computer capability, etc.). Representative "highest risk" compounds may be selected on the basis of: quantities present in EFPC; extent of environmental contamination, toxicity, or hazard; and mobility and persistence of the chemical in the environment. Reducing the number of chemicals considered in the risk assessment is specified as optional by EPA and is suggested as a device for focusing and facilitating the risk assessment process (EPA 1989a).

Three primary sources of information were used in identifying COCs as the focus of the BRA: a concentration-toxicity scoring system recommended by EPA, comparison with background, and the results of the Tier I screening-level risk assessment.

The concentration-toxicity scoring system is recommended by EPA in RAGS Vol. 1, Part A (EPA 1989a). This system uses measured concentrations and established toxicity measures to rank chemicals for the purposes of risk assessment without making quantitative assumptions about exposures and dose. Chemicals were divided into four groups based on the available toxicity measures: those exhibiting carcinogenic effects [chemicals for which oral cancer slope factors (CSFs) were available], those with systemic toxicity or noncarcinogenic effects [chemicals with oral reference doses (RfDs)], radionuclides [radionuclides for which oral CSFs were available in HEAST Fiscal Year 92 (EPA 1992e)], and chemicals for which toxicity measures were not available. For radionuclides, the slope factors used in the screening process take into account radionuclide progeny (i.e., decay products).

A toxicity score was calculated for each chemical in the first three groups. For radionuclides and carcinogenic effects of chemical contaminants, the score was determined by multiplying the concentration and the oral CSF. Noncarcinogenic effects were scored using the concentration divided by the RfD. Within each group, each chemical was ranked according to its percentage of the total score for the group. Those compounds that collectively represented 99% of the toxicity score for that group were considered COCs.

The arithmetic mean concentrations of all chemicals detected at EFPC were used to calculate the toxicity scores. Toxicity scores were then computed a second time using the maximum concentrations. The latter scores did not greatly change the rankings, but generally fewer contaminants accounted for 99% of the score. Toxicity scores based on the mean concentrations were used because this ranking included more chemicals as COCs, producing the most conservative approach and reducing the possibility of eliminating significant chemicals from the assessment.

The presence of the chemical in reference site (background) samples was also a factor used in determining the COCs. An analysis was performed to determine if differences in the mean site concentrations and the mean background concentrations were statistically significant. If these differences were not statistically significant and the toxicity score for that chemical was less than 1% of the total, the chemical was eliminated from consideration.

Additional factors other than the toxicity scores and comparison with background samples were considered in determining the COCs. These factors included the frequency of occurrence of a chemical in the samples tested, mobility and persistence, professional judgment based on knowledge of the processes used at the Y-12 Plant, and applicable or relevant and appropriate requirements (ARARs) [i.e., the maximum contaminant levels (MCLs) established under the Safe Drinking Water Act (SDWA) and federal ambient water quality criteria for the protection of

freshwater organisms]. Chemicals that were detected in less than 5% of the samples tested were not designated as COCs (EPA 1989a). Some radionuclides that were analyzed only in the Phase Ib analyses were listed as COCs in soils and sediments regardless of screening results because they are used in processes at the Y-12 Plant. These radionuclides include thorium-228, -230, -232, neptunium-237, and protactinium-233.

Contaminants that were not detected in any sample in a given media were eliminated from consideration. These analytes are listed in Appendix L along with the minimum, maximum, and average detection limits. Nutrient substances (e.g., sodium, potassium, calcium, magnesium, and iron) also were eliminated from consideration.

Environmental monitoring data from other studies of EFPC were not used in this analysis. All of the EFPC RI Phase Ia data used in the scoring have been obtained and analyzed following a consistent set of procedures, as discussed in Sect. 3.3. The quality of the analytical data was well-documented under EPA Level IV protocols. All sampling locations were surveyed and can be resampled, if necessary.

Toxicity scores for each chemical detected are listed by media in the tables in Sects. 5.1.2.1 through 5.1.2.5. Chemicals considered COCs based on the criteria listed above are denoted by an asterisk. The column labeled "EFPC proportion detected" represents the number of results greater than the detection limit over the total number of samples analyzed. The "Average EFPC result" is the arithmetic mean of the results. Results less than detection were set to one-half of the detection limit (except for radionuclides) and included in the calculation of the average concentration. EPA toxicity measures (CSFs and chronic RfDs) were taken from a summary of toxicity measures distributed to Oak Ridge contractors by the Biomedical and Environmental Information Analysis Section of the Health and Safety Research Division at ORNL [ORNL compiled the toxicity measures from the Integrated Risk Information System (IRIS) (EPA 1993) and HEAST (EPA 1992e)]. The mean concentration and toxicity score for the reference site at Hinds Creek also are listed where data were available. The radionuclide activity uncertainty is a pooled estimate of the uncertainty of the mean and is defined as the square root of uncertainty for each individual measurement divided by the total number of measurements.

5.1.2.1 Soil contaminants of concern

Metals:

antimony, arsenic, barium, beryllium, cadmium, chromium, copper, lead, manganese, mercury, nickel, selenium, silver, and zinc

Polyaromatic Hydrocarbons (PAHs):

benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, and indeno(1,2,3-cd)pyrene

PCBs:

Aroclor-1254 and Aroclor-1260

Radionuclides:

cesium-137, cobalt-60, neptunium-237, protactinium-233, thorium-228, thorium-230, thorium-232, uranium-234, uranium-235, and uranium-238.

Hazard Score - Noncarcinogenic Effects

Mercury accounts for 65% of the total toxicity (Table 5.1), based on the score for noncarcinogenic effects. Another metal that contributes significantly (15%) to the total toxicity score is arsenic. Thallium was eliminated from consideration because it was infrequently detected. Vanadium also was eliminated because the average concentration at EFPC was less than the background concentration.

Although no toxicity values are available for lead, it has been included as a COC. EPA recommends that quantitative estimates of risk associated with lead be based on an alternative approach in which blood lead uptake is compared to the latest Center for Disease Control guideline of 10 μg/dL blood lead in children. These blood lead levels can be projected by using a pharmokinetic lead uptake model (LEAD 0.60, EPA Lead Uptake/Biokinetic Model). The EFPC BRA addresses lead toxicity separately using this model.

The organic compounds in Table 5.1 together represent less than 1% of total noncancer toxicity. These compounds were not included as COCs based on the noncancer scoring.

Hazard Score - Carcinogenic Effects

PAHs account for a significant percentage of the carcinogenic risk scoring in soil (approximately 22%). Seven carcinogenic PAHs have been included as COCs. The scoring of carcinogenic PAHs was conducted using the Region IV toxicity equivalence factors (TEFs) (EPA 1992a) and the very conservative CSF of 11.5 for benzo(a)pyrene. This value currently has been revised downward by EPA to approximately 7.

Two PCBs, Aroclor-1254 and Aroclor-1260, also were detected in the soils and are considered COCs. Pentachlorophenol is not included as a COC because of its low frequency of

Table 5.1. East Fork Poplar Creek Phase Ia and Ib results: toxicity scores based on average contaminant concentrations in EFPC soil ranked by toxicity

PARAMETER	EFPC Proportion Detected	Mean EFPC Result	Measurement Units	EFPC Toxicity Score	EFPC % of Total Toxicity	Toxicity Factor	Background Proportion Detected	Mean BKGD Result	BKGD Toxicity Score	Radiation Measurement Uncertainty
Noncarcinogenic Score										
*Mercury	510/1562	33.40	MG/KG	1.11E+05	65.37	3.00E-04	1/ 12	0.06	2.15E+02	.
*Arsenic	1150/1162	7.70	MG/KG	2.57E+04	15.07	3.00E-04	12/ 12	2.54	8.47E+03	.
Thallium	1/ 89	0.57	MG/KG	7.06E+03	4.15	8.00E-05	0/ 12	0.25	3.10E+03	.
*Manganese	93/ 93	932.00	MG/KG	6.66E+03	3.91	1.40E-01	12/ 12	509.00	3.64E+03	.
*Cadmium	65/ 93	4.23	MG/KG	4.23E+03	2.48	1.00E-03	0/ 12	0.37	3.72E+02	.
Vanadium	93/ 93	24.00	MG/KG	3.43E+03	2.01	7.00E-03	12/ 12	19.50	2.78E+03	.
*Copper	92/ 93	80.30	MG/KG	2.17E+03	1.27	3.70E-02	12/ 12	8.65	2.34E+02	.
*Antimony	1254/1455	0.82	MG/KG	2.04E+03	1.20	4.00E-04	0/ 7	3.74	9.36E+03	.
*Selenium	382/1540	9.82	MG/KG	1.96E+03	1.15	5.00E-03	0/ 12	0.37	7.43E+01	.
*Nickel	91/ 93	36.20	MG/KG	1.81E+03	1.06	2.00E-02	12/ 12	13.90	6.97E+02	.
*Barium	93/ 93	124.00	MG/KG	1.77E+03	1.04	7.00E-02	12/ 12	112.00	1.60E+03	.
*Silver	38/ 93	3.69	MG/KG	7.38E+02	0.43	5.00E-03	0/ 12	0.87	1.74E+02	.
Heptachlor Epoxide	7/ 89	3.21	UG/KG	2.47E+02	0.15	1.30E-05	0/ 11	4.93	3.79E+02	.
*Zinc	393/1522	71.90	MG/KG	2.40E+02	0.14	3.00E-01	11/ 12	33.30	1.11E+02	.
Dieldrin	9/ 89	8.74	UG/KG	1.75E+02	0.10	5.00E-05	0/ 11	9.86	1.97E+02	.
*Beryllium	68/ 93	0.79	MG/KG	1.59E+02	0.09	5.00E-03	12/ 12	0.68	1.35E+02	.
Cyanide	16/ 36	2.95	MG/KG	1.47E+02	0.09	2.00E-02	0/ 11	0.31	1.55E+01	.
Aldrin	9/ 89	3.11	UG/KG	1.04E+02	0.06	3.00E-05	0/ 11	4.93	1.64E+02	.
*Chromium	1585/1585	64.80	MG/KG	6.48E+01	0.04	1.00E+00	12/ 12	20.20	2.02E+01	.
Pentachlorophenol	2/ 86	801.00	UG/KG	2.67E+01	0.02	3.00E-02	0/ 11	1000.00	3.33E+01	.
Pyrene	71/ 86	676.00	UG/KG	2.25E+01	0.01	3.00E-02	0/ 11	205.00	6.85E+00	.
*Benzo(b)fluoranthene	70/ 86	657.00	UG/KG	2.19E+01	0.01	3.00E-02	0/ 11	205.00	6.85E+00	.
Fluoranthene	71/ 86	872.00	UG/KG	2.18E+01	0.01	4.00E-02	0/ 11	205.00	5.14E+00	.
Endrin	8/ 89	6.26	UG/KG	2.09E+01	0.01	3.00E-04	0/ 11	9.86	3.29E+01	.
*Benzo(k)fluoranthene	64/ 86	608.00	UG/KG	2.03E+01	0.01	3.00E-02	0/ 11	205.00	6.85E+00	.
Phenanthrene	57/ 86	509.00	UG/KG	1.70E+01	0.01	3.00E-02	0/ 11	205.00	6.85E+00	.
*Chrysene	60/ 86	461.00	UG/KG	1.54E+01	0.01	3.00E-02	0/ 11	205.00	6.85E+00	.
*Benzo(a)anthracene	60/ 86	453.00	UG/KG	1.51E+01	0.01	3.00E-02	0/ 11	205.00	6.85E+00	.
*Benzo(a)pyrene	57/ 86	427.00	UG/KG	1.42E+01	0.01	3.00E-02	0/ 11	205.00	6.85E+00	.
4,4'-DDT	4/ 89	6.38	UG/KG	1.28E+01	0.01	5.00E-04	0/ 11	9.86	1.97E+01	.
Di-n-octylphthalate	2/ 86	235.00	UG/KG	1.18E+01	0.01	2.00E-02	0/ 11	205.00	1.03E+01	.
Benzo(g,h,i)perylene	39/ 86	333.00	UG/KG	1.11E+01	0.01	3.00E-02	0/ 11	205.00	6.85E+00	.
*Indeno(1,2,3-c,d)pyrene	44/ 86	329.00	UG/KG	1.10E+01	0.01	3.00E-02	0/ 11	205.00	6.85E+00	.
gamma-BHC (Lindane)	3/ 89	3.27	UG/KG	1.09E+01	0.01	3.00E-04	0/ 11	4.93	1.64E+01	.
bis(2-Ethylhexyl)phthalate	39/ 86	189.00	UG/KG	9.47E+00	0.01	2.00E-02	2/ 11	247.00	1.24E+01	.
*Dibenzo(a,h)anthracene	23/ 86	220.00	UG/KG	7.32E+00	0.00	3.00E-02	0/ 11	205.00	6.85E+00	.
Acenaphthylene	10/ 86	217.00	UG/KG	7.24E+00	0.00	3.00E-02	0/ 11	205.00	6.85E+00	.
2-Methylnaphthalene	24/ 86	213.00	UG/KG	7.09E+00	0.00	3.00E-02	0/ 11	205.00	6.85E+00	.
Heptachlor	17/ 89	3.02	UG/KG	6.04E+00	0.00	5.00E-04	0/ 11	4.93	9.85E+00	.
Fluorene	11/ 86	239.00	UG/KG	5.99E+00	0.00	4.00E-02	0/ 11	205.00	5.14E+00	.
Acenaphthene	9/ 86	250.00	UG/KG	4.16E+00	0.00	6.00E-02	0/ 11	205.00	3.42E+00	.
Di-n-butylphthalate	11/ 86	231.00	UG/KG	2.31E+00	0.00	1.00E-01	0/ 11	205.00	2.05E+00	.
Acetone	1/ 12	218.00	UG/KG	2.18E+00	0.00	1.00E-01	./ .	.	.	.
Butylbenzylphthalate	7/ 86	225.00	UG/KG	1.12E+00	0.00	2.00E-01	0/ 11	205.00	1.03E+00	.
Anthracene	28/ 86	229.00	UG/KG	7.64E-01	0.00	3.00E-01	0/ 11	205.00	6.85E-01	.
Diethylphthalate	1/ 86	370.00	UG/KG	4.62E-01	0.00	8.00E-01	0/ 11	205.00	2.57E-01	.
Benzoic Acid	2/ 30	1170.00	UG/KG	2.93E-01	0.00	4.00E+00	2/ 11	862.00	2.16E-01	.

* Contaminant of Concern

Table 5.1. (continued)

PARAMETER	EFPC Proportion Detected	Mean EFPC Result	Measurement Units	EFPC Toxicity Score	EFPC % of Total Toxicity	Toxicity Factor	Background Proportion Detected	Mean BKGD Result	BKGD Toxicity Score	Radiation Measurement Uncertainty
Carcinogenic Score										
*Arsenic	1150/1162	7.70	MG/KG	1.35E+01	48.31	1.75E+00	12/ 12	2.54	4.45E+00	.
*Beryllium	68/ 93	0.79	MG/KG	3.41E+00	12.23	4.30E+00	12/ 12	0.68	2.90E+00	.
*Aroclor-1260	76/ 109	421.00	UG/KG	3.24E+00	11.62	7.70E+00	0/ 11	98.60	7.60E-01	.
*Benzo(a)pyrene	57/ 86	427.00	UG/KG	3.12E+00	11.17	7.30E+00	0/ 11	205.00	1.50E+00	.
*Dibenzo(a,h)anthracene	23/ 86	220.00	UG/KG	1.60E+00	5.74	7.30E+00	0/ 11	205.00	1.50E+00	.
*Aroclor-1254	11/ 109	153.00	UG/KG	1.18E+00	4.22	7.70E+00	0/ 11	98.60	7.60E-01	.
*Benzo(b)fluoranthene	70/ 86	657.00	UG/KG	4.79E-01	1.72	7.30E-01	0/ 11	205.00	1.50E-01	.
*Benzo(k)fluoranthene	64/ 86	608.00	UG/KG	4.44E-01	1.59	7.30E-01	0/ 11	205.00	1.50E-01	.
*Benzo(a)anthracene	60/ 86	453.00	UG/KG	3.31E-01	1.19	7.30E-01	0/ 11	205.00	1.50E-01	.
*Indeno(1,2,3-c,d)pyrene	44/ 86	329.00	UG/KG	2.40E-01	0.86	7.30E-01	0/ 11	205.00	1.50E-01	.
Dieldrin	9/ 89	8.74	UG/KG	1.40E-01	0.50	1.60E+01	0/ 11	9.86	1.58E-01	.
Pentachlorophenol	2/ 86	801.00	UG/KG	9.61E-02	0.34	1.20E-01	0/ 11	1000.00	1.20E-01	.
Aldrin	9/ 89	3.11	UG/KG	5.29E-02	0.19	1.70E+01	0/ 11	4.93	8.38E-02	.
*Chrysene	60/ 86	461.00	UG/KG	3.37E-02	0.12	7.30E-02	0/ 11	205.00	1.50E-02	.
Heptachlor Epoxide	7/ 89	3.21	UG/KG	2.92E-02	0.10	9.10E+00	0/ 11	4.93	4.48E-02	.
Heptachlor	17/ 89	3.02	UG/KG	1.36E-02	0.05	4.50E+00	0/ 11	4.93	2.22E-02	.
gamma-BHC (Lindane)	3/ 89	3.27	UG/KG	4.25E-03	0.02	1.30E+00	0/ 11	4.93	6.41E-03	.
Carbazole	11/ 56	191.00	UG/KG	3.82E-03	0.01	2.00E-02	./ .	.	.	.
bis(2-Ethylhexyl)phthalate	39/ 86	189.00	UG/KG	2.65E-03	0.01	1.40E-02	2/ 11	247.00	3.46E-03	.
4,4'-DDT	4/ 89	6.38	UG/KG	2.17E-03	0.01	3.40E-01	0/ 11	9.86	3.35E-03	.
4,4'-DDE	32/ 88	5.75	UG/KG	1.95E-03	0.01	3.40E-01	0/ 11	9.86	3.35E-03	.
4,4'-DDD	6/ 89	6.24	UG/KG	1.50E-03	0.01	2.40E-01	0/ 11	9.86	2.37E-03	.
Radionuclide Score										
*Uranium-238	1312/1555	3.84	PCI/G	1.08E-10	28.91	2.80E-11	9/ 9	0.21	6.00E-12	0.01670
*Thorium-228	22/ 22	1.77	PCI/G	9.72E-11	26.13	5.50E-11	./ .	.	.	0.03610
*Uranium-234	1299/1542	3.70	PCI/G	5.92E-11	15.92	1.60E-11	9/ 9	0.26	4.23E-12	0.01800
*Thorium-230	16/ 16	4.02	PCI/G	5.23E-11	14.04	1.30E-11	./ .	.	.	0.06270
*Cesium-137	100/ 108	0.98	PCI/G	2.73E-11	7.33	2.80E-11	11/ 12	0.37	1.04E-11	0.01940
*Thorium-232	22/ 22	1.24	PCI/G	1.49E-11	4.00	1.20E-11	./ .	.	.	0.03070
*Neptunium-237	14/ 75	0.03	PCI/G	5.67E-12	1.52	2.20E-10	1/ 12	0.01	1.47E-12	0.01290
Americium-241	1/ 49	0.02	PCI/G	4.97E-12	1.33	2.40E-10	0/ 12	0.00	1.54E-13	0.07540
*Uranium-235	1648/1649	0.17	PCI/G	2.77E-12	0.74	1.60E-11	9/ 9	0.01	2.31E-13	0.00104
*Cobalt-60	17/ 106	0.02	PCI/G	2.40E-13	0.06	1.50E-11	2/ 12	0.00	1.01E-14	0.00679
*Protactinium-233	3/ 53	0.01	PCI/G	1.38E-14	0.00	1.00E-12	./ .	.	.	0.02970

* Contaminant of Concern

Table 5.1. (continued)

PARAMETER	EFPC Proportion Detected	Mean EFPC Result	Measurement Units	EFPC Toxicity Score	EFPC % of Total Toxicity	Toxicity Factor	Background Proportion Detected	Mean BKGD Result	BKGD Toxicity Score	Radiation Measurement Uncertainty
EPA Risk Factor = None Available										
4-Chloro-3-methylphenol	2/ 86	235.00	UG/KG	.	.	.	0/ 11	205.00	.	.
4-Nitrophenol	1/ 86	813.00	UG/KG	.	.	.	0/ 11	1000.00	.	.
Aluminum	93/ 93	13800.00	MG/KG	.	.	.	12/ 12	12400.00	.	.
Cobalt	89/ 93	12.40	MG/KG	.	.	.	12/ 12	9.21	.	.
Dibenzofuran	8/ 86	234.00	UG/KG	.	.	.	0/ 11	205.00	.	.
Endosulfan I	2/ 89	3.18	UG/KG	.	.	.	0/ 11	4.93	.	.
Endosulfan II	4/ 89	6.28	UG/KG	.	.	.	0/ 11	9.86	.	.
Endosulfan Sulfate	1/ 89	6.35	UG/KG	.	.	.	0/ 11	9.86	.	.
Endrin Aldehyde	4/ 56	2.16	UG/KG	.	.	.	./ .	.	.	.
Endrin Ketone	1/ 89	6.34	UG/KG	.	.	.	0/ 11	9.86	.	.
Lead	90/ 90	49.10	MG/KG	.	.	.	12/ 12	12.60	.	.
Naphthalene	16/ 86	237.00	UG/KG	.	.	.	0/ 11	205.00	.	.
alpha-Chlordane	35/ 89	26.00	UG/KG	.	.	.	0/ 11	49.30	.	.
delta-BHC	4/ 89	3.16	UG/KG	.	.	.	0/ 11	4.93	.	.
gamma-Chlordane	30/ 89	25.60	UG/KG	.	.	.	0/ 11	49.30	.	.
p,p'-Methoxychlor	6/ 89	31.40	UG/KG	.	.	.	0/ 11	49.30	.	.

EPA Risk Factors were taken from the IRIS and HEAST Tables as updated through February, 1993.
Results less than detection were set to 1/2 DL (except for radionuclides).

detection. Bis(2-ethylhexyl)phthalate, a common laboratory contaminant, was detected but represented only 0.01% of the cancer risk.

Arsenic and beryllium, two potential carcinogens, are considered COCs. They account for 48% and 12% of the total toxicity for carcinogens, respectively.

Hazard Score - Radionuclides

Generally, uranium isotope counts were one order of magnitude greater in EFPC soils than the background site. Cesium measurements also were slightly elevated in EFPC soils. Since americium-241 counts were not significantly different from the background site and the measurement uncertainty was relatively high, they will not be considered further. Thorium-228, -230, -232, neptunium-237, and protactinium-233 were included in the list of COCs because they are part of processes at the Y-12 Plant.

Chemicals with no Score

Two metals and two organic compounds were detected but could not be scored, since toxicity measures were not available. Aluminum and cobalt were detected in EFPC at concentrations that were not statistically different from the background site (t-test on mean values). 4-Nitrophenol and 4-chloro-3-methylphenol were detected infrequently in EFPC, and are not considered part of processes at the Y-12 Plant. Therefore, they are not addressed in the evaluation.

5.1.2.2 Sediment contaminants of concern

Metals:

arsenic, barium, beryllium, cadmium, chromium, copper, lead, manganese, mercury, nickel, vanadium, and zinc

PAHs:

benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, and indeno(1,2,3-cd)pyrene

Pesticides:

dieldrin

PCBs:

Aroclor-1254 and Aroclor-1260.

Hazard Score - Noncarcinogenic Effects

Mercury accounts for 61% of the total toxicity (Table 5.2) based on the score for noncarcinogenic effects. Other metals that are largely responsible for the score are arsenic, manganese, and vanadium. Again, as noted previously, lead was not removed from consideration based on the screening results, and is therefore considered a COC.

The organic compounds in Table 5.2 together represent less than 1% of total noncancer toxicity. The compounds were not included as COCs based on the noncancer scoring.

Hazard Score - Carcinogenic Effects

Arsenic and beryllium are responsible for the majority (64%) of the total toxicity for carcinogens. PAHs are also responsible for the carcinogenic risk from sediments. Seven PAHs are included as COCs. PCBs also were detected in the sediments and are considered COCs. Several pesticides (alpha-BHC, gamma-BHC, heptachlor epoxide, 4,4'-DDD, 4,4'-DDE, and 4,4'-DDT) are not included as COCs because many were infrequently detected, and together they comprise less than 1% of the carcinogenic risk. Bis(2-ethylhexyl)phthalate was detected, but represented only 0.02% of the cancer risk.

Chemicals with no Score

Two metals were detected but could not be scored, since toxicity measures were not available. Aluminum and cobalt were detected in EFPC at concentrations that were not statistically different from the background site (t-test on mean values).

5.1.2.3 Groundwater contaminants of concern

Metals:

arsenic, barium, beryllium, chromium, copper, lead, manganese, mercury, nickel, and vanadium

Volatiles:

acetone and methylene chloride

Radionuclides:

total radium, uranium-234, and uranium-238.

PAHs and PCBs were infrequently detected and are not included in the list of COCs (Table 5.3). Total radium was included on the list because it was measured at significant levels

Table 5.2. East Fork Poplar Creek Phase Ia and Ib results: toxicity scores based on average contaminant concentrations in EFPC sediment ranked by toxicity

PARAMETER	EFPC Proportion Detected	Mean EFPC Result	Measurement Units	EFPC Toxicity Score	EFPC % of Total Toxicity	Toxicity Factor	Background Proportion Detected	Mean BKGD Result	BKGD Toxicity Score	Radiation Measurement Uncertainty
Noncarcinogenic Score										
*Mercury	35/ 35	15.00	MG/KG	4.98E+04	61.30	3.00E-04	0/ 2	0.06	2.00E+02	.
*Arsenic	35/ 35	4.55	MG/KG	1.52E+04	18.67	3.00E-04	2/ 2	4.25	1.42E+04	.
*Manganese	35/ 35	886.00	MG/KG	6.33E+03	7.79	1.40E-01	2/ 2	631.00	4.51E+03	.
*Vanadium	35/ 35	29.00	MG/KG	4.15E+03	5.10	7.00E-03	2/ 2	28.50	4.06E+03	.
*Cadmium	15/ 35	1.42	MG/KG	1.42E+03	1.74	1.00E-03	0/ 2	0.55	5.50E+02	.
*Nickel	35/ 35	23.10	MG/KG	1.16E+03	1.42	2.00E-02	2/ 2	23.40	1.17E+03	.
*Barium	35/ 35	78.10	MG/KG	1.12E+03	1.37	7.00E-02	2/ 2	167.00	2.38E+03	.
*Copper	35/ 35	24.00	MG/KG	6.48E+02	0.80	3.70E-02	2/ 2	14.20	3.84E+02	.
*Zinc	35/ 35	97.10	MG/KG	3.24E+02	0.40	3.00E-01	2/ 2	54.80	1.83E+02	.
Heptachlor Epoxide	1/ 34	4.04	UG/KG	3.11E+02	0.38	1.30E-05	0/ 2	5.25	4.04E+02	.
*Beryllium	29/ 35	0.92	MG/KG	1.83E+02	0.23	5.00E-03	0/ 2	0.47	9.35E+01	.
*Dieldrin	8/ 34	7.68	UG/KG	1.54E+02	0.19	5.00E-05	0/ 2	10.80	2.15E+02	.
Aldrin	14/ 34	3.71	UG/KG	1.24E+02	0.15	3.00E-05	0/ 2	5.25	1.75E+02	.
Selenium	2/ 33	0.56	MG/KG	1.12E+02	0.14	5.00E-03	0/ 2	0.40	7.95E+01	.
*Chromium	35/ 35	47.70	MG/KG	4.77E+01	0.06	1.00E+00	2/ 2	28.80	2.88E+01	.
Endrin	11/ 34	7.54	UG/KG	2.51E+01	0.03	3.00E-04	0/ 2	10.80	3.58E+01	.
Pyrene	17/ 33	473.00	UG/KG	1.58E+01	0.02	3.00E-02	0/ 2	220.00	7.33E+00	.
4,4'-DDT	7/ 34	7.82	UG/KG	1.56E+01	0.02	5.00E-04	0/ 2	10.80	2.15E+01	.
gamma-BHC (Lindane)	3/ 34	4.01	UG/KG	1.34E+01	0.02	3.00E-04	0/ 2	5.25	1.75E+01	.
*Benzo(b)fluoranthene	15/ 33	375.00	UG/KG	1.25E+01	0.02	3.00E-02	0/ 2	220.00	7.33E+00	.
Fluoranthene	18/ 33	499.00	UG/KG	1.25E+01	0.02	4.00E-02	0/ 2	220.00	5.50E+00	.
Di-n-octylphthalate	1/ 33	226.00	UG/KG	1.13E+01	0.01	2.00E-02	0/ 2	220.00	1.10E+01	.
*Benzo(a)anthracene	14/ 33	325.00	UG/KG	1.08E+01	0.01	3.00E-02	0/ 2	220.00	7.33E+00	.
*Benzo(a)pyrene	11/ 33	315.00	UG/KG	1.05E+01	0.01	3.00E-02	0/ 2	220.00	7.33E+00	.
*Chrysene	15/ 33	309.00	UG/KG	1.03E+01	0.01	3.00E-02	0/ 2	220.00	7.33E+00	.
*Benzo(k)fluoranthene	12/ 33	307.00	UG/KG	1.02E+01	0.01	3.00E-02	0/ 2	220.00	7.33E+00	.
bis(2-Ethylhexyl)phthalate	15/ 33	203.00	UG/KG	1.01E+01	0.01	2.00E-02	0/ 2	220.00	1.10E+01	.
Phenanthrene	11/ 33	296.00	UG/KG	9.86E+00	0.01	3.00E-02	0/ 2	220.00	7.33E+00	.
*Indeno(1,2,3-c,d)pyrene	7/ 33	276.00	UG/KG	9.18E+00	0.01	3.00E-02	0/ 2	220.00	7.33E+00	.
Benzo(g,h,i)perylene	6/ 33	271.00	UG/KG	9.04E+00	0.01	3.00E-02	0/ 2	220.00	7.33E+00	.
Heptachlor	11/ 34	3.75	UG/KG	7.50E+00	0.01	5.00E-04	0/ 2	5.25	1.05E+01	.
*Dibenzo(a,h)anthracene	3/ 33	219.00	UG/KG	7.32E+00	0.01	3.00E-02	0/ 2	220.00	7.33E+00	.
Acenaphthylene	1/ 33	216.00	UG/KG	7.19E+00	0.01	3.00E-02	0/ 2	220.00	7.33E+00	.
Fluorene	3/ 33	207.00	UG/KG	5.17E+00	0.01	4.00E-02	0/ 2	220.00	5.50E+00	.
Acenaphthene	1/ 33	220.00	UG/KG	3.67E+00	0.00	6.00E-02	0/ 2	220.00	3.67E+00	.
Di-n-butylphthalate	3/ 33	241.00	UG/KG	2.41E+00	0.00	1.00E-01	0/ 2	220.00	2.20E+00	.
Butylbenzylphthalate	1/ 33	225.00	UG/KG	1.13E+00	0.00	2.00E-01	0/ 2	220.00	1.10E+00	.
Anthracene	7/ 33	216.00	UG/KG	7.19E-01	0.00	3.00E-01	0/ 2	220.00	7.33E-01	.
Diethylphthalate	1/ 33	215.00	UG/KG	2.69E-01	0.00	8.00E-01	0/ 2	220.00	2.75E-01	.
Benzoic Acid	1/ 6	922.00	UG/KG	2.30E-01	0.00	4.00E+00	0/ 2	1080.00	2.69E-01	.
Dimethylphthalate	0/ 33	225.00	UG/KG	2.25E-02	0.00	1.00E+01	0/ 2	220.00	2.20E-02	.

* Contaminant of Concern

Table 5.2. (continued)

PARAMETER	EFPC Proportion Detected	Mean EFPC Result	Measurement Units	EFPC Toxicity Score	EFPC % of Total Toxicity	Toxicity Factor	Background Proportion Detected	Mean BKGD Result	BKGD Toxicity Score	Radiation Measurement Uncertainty
Carcinogenic Score										
*Arsenic	35/ 35	4.55	MG/KG	7.97E+00	43.14	1.75E+00	2/ 2	4.25	7.44E+00	.
*Beryllium	29/ 35	0.92	MG/KG	3.94E+00	21.30	4.30E+00	0/ 2	0.47	2.01E+00	.
*Benzo(a)pyrene	11/ 33	315.00	UG/KG	2.30E+00	12.46	7.30E+00	0/ 2	220.00	1.61E+00	.
*Dibenzo(a,h)anthracene	3/ 33	219.00	UG/KG	1.60E+00	8.67	7.30E+00	0/ 2	220.00	1.61E+00	.
*Aroclor-1254	10/ 34	103.00	UG/KG	7.94E-01	4.30	7.70E+00	0/ 2	108.00	8.28E-01	.
*Aroclor-1260	2/ 34	80.80	UG/KG	6.22E-01	3.37	7.70E+00	0/ 2	108.00	8.28E-01	.
*Benzo(b)fluoranthene	15/ 33	375.00	UG/KG	2.74E-01	1.48	7.30E-01	0/ 2	220.00	1.61E-01	.
*Benzo(a)anthracene	14/ 33	325.00	UG/KG	2.37E-01	1.29	7.30E-01	0/ 2	220.00	1.61E-01	.
*Benzo(k)fluoranthene	12/ 33	307.00	UG/KG	2.24E-01	1.21	7.30E-01	0/ 2	220.00	1.61E-01	.
*Indeno(1,2,3-c,d)pyrene	7/ 33	276.00	UG/KG	2.01E-01	1.09	7.30E-01	0/ 2	220.00	1.61E-01	.
*Dieldrin	8/ 34	7.68	UG/KG	1.23E-01	0.66	1.60E+01	0/ 2	10.80	1.72E-01	.
Aldrin	14/ 34	3.71	UG/KG	6.30E-02	0.34	1.70E+01	0/ 2	5.25	8.93E-02	.
Heptachlor Epoxide	1/ 34	4.04	UG/KG	3.68E-02	0.20	9.10E+00	0/ 2	5.25	4.78E-02	.
alpha-BHC	2/ 34	4.01	UG/KG	2.52E-02	0.14	6.30E+00	0/ 2	5.25	3.31E-02	.
*Chrysene	15/ 33	309.00	UG/KG	2.26E-02	0.12	7.30E-02	0/ 2	220.00	1.61E-02	.
Heptachlor	11/ 34	3.75	UG/KG	1.69E-02	0.09	4.50E+00	0/ 2	5.25	2.36E-02	.
beta-BHC	4/ 34	4.01	UG/KG	7.22E-03	0.04	1.80E+00	0/ 2	5.25	9.45E-03	.
gamma-BHC (Lindane)	3/ 34	4.01	UG/KG	5.21E-03	0.03	1.30E+00	0/ 2	5.25	6.83E-03	.
Carbazole	3/ 27	200.00	UG/KG	4.00E-03	0.02	2.00E-02	./ .	.	.	.
bis(2-Ethylhexyl)phthalate	15/ 33	203.00	UG/KG	2.84E-03	0.02	1.40E-02	0/ 2	220.00	3.08E-03	.
4,4'-DDE	2/ 34	7.96	UG/KG	2.71E-03	0.01	3.40E-01	0/ 2	10.80	3.66E-03	.
4,4'-DDT	7/ 34	7.82	UG/KG	2.66E-03	0.01	3.40E-01	0/ 2	10.80	3.66E-03	.
4,4'-DDD	7/ 34	7.83	UG/KG	1.88E-03	0.01	2.40E-01	0/ 2	10.80	2.58E-03	.
Radionuclide Score										
Uranium-238	19/ 19	3.24	PCI/G	9.06E-11	32.03	2.80E-11	2/ 2	0.17	4.76E-12	0.04920
Uranium-234	19/ 19	4.23	PCI/G	6.77E-11	23.92	1.60E-11	2/ 2	0.23	3.68E-12	0.05590
Thorium-228	2/ 2	0.86	PCI/G	4.75E-11	16.79	5.50E-11	./ .	.	.	0.08650
Neptunium-237	3/ 20	0.18	PCI/G	3.91E-11	13.81	2.20E-10	0/ 2	0.01	1.42E-12	0.02270
Cesium-137	35/ 35	0.56	PCI/G	1.57E-11	5.56	2.80E-11	1/ 2	0.04	1.11E-12	0.01720
Thorium-230	1/ 1	0.79	PCI/G	1.02E-11	3.62	1.30E-11	./ .	.	.	0.10100
Thorium-232	2/ 2	0.64	PCI/G	7.66E-12	2.71	1.20E-11	./ .	.	.	0.07040
Uranium-235	16/ 16	0.25	PCI/G	3.94E-12	1.39	1.60E-11	2/ 2	0.01	1.60E-13	0.01560
Cobalt-60	12/ 35	0.03	PCI/G	4.33E-13	0.15	1.50E-11	0/ 2	-0.01	0.00E+00	0.01220
Protactinium-233	2/ 27	0.04	PCI/G	4.10E-14	0.01	1.00E-12	./ .	.	.	0.03130

* Contaminant of Concern

Table 5.2. (continued)

PARAMETER	EFPC Proportion Detected	Mean EFPC Result	Measurement Units	EFPC Toxicity Score	EFPC % of Total Toxicity	Toxicity Factor	Background Proportion Detected	Mean BKGD Result	BKGD Toxicity Score	Radiation Measurement Uncertainty
EPA Risk Factor = None Available										
Aluminum	35/ 35	8030.00	MG/KG	.	.	.	2/ 2	13700.00	.	.
Cobalt	35/ 35	13.00	MG/KG	.	.	.	2/ 2	12.40	.	.
Dibenzofuran	0/ 33	225.00	UG/KG	.	.	.	0/ 2	220.00	.	.
Endosulfan I	1/ 34	4.06	UG/KG	.	.	.	0/ 2	5.25	.	.
Endosulfan II	3/ 34	7.94	UG/KG	.	.	.	0/ 2	10.80	.	.
Endosulfan Sulfate	6/ 34	7.72	UG/KG	.	.	.	0/ 2	10.80	.	.
Endrin Aldehyde	12/ 27	1.82	UG/KG	.	.	.	./ .	.	.	.
Lead	35/ 35	53.00	MG/KG	.	.	.	2/ 2	6.15	.	.
Naphthalene	0/ 33	219.00	UG/KG	.	.	.	0/ 2	220.00	.	.
alpha-Chlordane	22/ 34	32.40	UG/KG	.	.	.	0/ 2	52.50	.	.
delta-BHC	2/ 34	4.09	UG/KG	.	.	.	0/ 2	5.25	.	.
gamma-Chlordane	23/ 34	32.70	UG/KG	.	.	.	0/ 2	52.50	.	.

EPA Risk Factors were taken from the IRIS and HEAST Tables as updated through February, 1993.
Results less than detection were set to 1/2 DL (except for radionuclides).

Table 5.3. East Fork Poplar Creek Phase Ia and Ib results: toxicity scores based on average contaminant concentrations in EFPC groundwater ranked by toxicity

PARAMETER	EFPC Proportion Detected	Mean EFPC Result	Measurement Units	EFPC Toxicity Score	EFPC % of Total Toxicity	Toxicity Factor	Background Proportion Detected	Mean BKGD Result	BKGD Toxicity Score	Radiation Measurement Uncertainty
				Noncarcinogenic Score						
*Manganese	161/ 166	3340.00	UG/L	6.69E+02	83.34	5.00E-03	./	.	.	.
Antimony	7/ 166	15.00	UG/L	3.74E+01	4.67	4.00E-04	./	.	.	.
*Mercury	61/ 141	8.39	UG/L	2.80E+01	3.49	3.00E-04	./	.	.	.
*Arsenic	42/ 162	7.20	UG/L	2.40E+01	2.99	3.00E-04	./	.	.	.
Thallium	1/ 166	1.24	UG/L	1.55E+01	1.93	8.00E-05	./	.	.	.
Cadmium	9/ 166	2.79	UG/L	5.59E+00	0.70	5.00E-04	./	.	.	.
*Barium	165/ 166	279.00	UG/L	3.99E+00	0.50	7.00E-02	./	.	.	.
*Acetone	24/ 79	363.00	UG/L	3.63E+00	0.45	1.00E-01	./	.	.	.
*Vanadium	62/ 166	22.10	UG/L	3.16E+00	0.39	7.00E-03	./	.	.	.
*Copper	75/ 166	85.50	UG/L	2.31E+00	0.29	3.70E-02	./	.	.	.
Heptachlor Epoxide	1/ 80	0.03	UG/L	1.98E+00	0.25	1.30E-05	./	.	.	.
*Nickel	72/ 166	29.30	UG/L	1.47E+00	0.18	2.00E-02	./	.	.	.
*Methylene Chloride	9/ 80	69.80	UG/L	1.16E+00	0.14	6.00E-02	./	.	.	.
4-Methylphenol	1/ 69	5.01	UG/L	1.00E+00	0.12	5.00E-03	./	.	.	.
Pentachlorophenol	1/ 68	17.40	UG/L	5.81E-01	0.07	3.00E-02	./	.	.	.
Tetrachloroethene	1/ 80	5.78	UG/L	5.78E-01	0.07	1.00E-02	./	.	.	.
Chloroform	2/ 80	5.76	UG/L	5.76E-01	0.07	1.00E-02	./	.	.	.
*Beryllium	18/ 166	1.27	UG/L	2.54E-01	0.03	5.00E-03	./	.	.	.
bis(2-Ethylhexyl)phthalate	11/ 77	5.08	UG/L	2.54E-01	0.03	2.00E-02	./	.	.	.
Cyanide	1/ 81	5.00	UG/L	2.50E-01	0.03	2.00E-02	./	.	.	.
Zinc	95/ 166	57.20	UG/L	1.91E-01	0.02	3.00E-01	./	.	.	.
Endrin	1/ 80	0.05	UG/L	1.71E-01	0.02	3.00E-04	./	.	.	.
2-Butanone	1/ 80	8.55	UG/L	1.71E-01	0.02	5.00E-02	./	.	.	.
Pyrene	1/ 77	5.06	UG/L	1.69E-01	0.02	3.00E-02	./	.	.	.
Benzo(a)anthracene	1/ 77	5.03	UG/L	1.68E-01	0.02	3.00E-02	./	.	.	.
Benzo(b)fluoranthene	1/ 77	5.03	UG/L	1.68E-01	0.02	3.00E-02	./	.	.	.
Chrysene	1/ 77	5.03	UG/L	1.68E-01	0.02	3.00E-02	./	.	.	.
Benzo(a)pyrene	1/ 77	5.02	UG/L	1.67E-01	0.02	3.00E-02	./	.	.	.
Benzo(k)fluoranthene	1/ 77	5.02	UG/L	1.67E-01	0.02	3.00E-02	./	.	.	.
Fluoranthene	1/ 77	5.05	UG/L	1.26E-01	0.02	4.00E-02	./	.	.	.
4,4'-DDT	1/ 80	0.05	UG/L	1.03E-01	0.01	5.00E-04	./	.	.	.
gamma-BHC (Lindane)	1/ 80	0.03	UG/L	8.56E-02	0.01	3.00E-04	./	.	.	.
Carbon Disulfide	6/ 80	5.76	UG/L	5.76E-02	0.01	1.00E-01	./	.	.	.
Di-n-butylphthalate	3/ 77	4.81	UG/L	4.81E-02	0.01	1.00E-01	./	.	.	.
Butylbenzylphthalate	1/ 77	5.01	UG/L	2.50E-02	0.00	2.00E-01	./	.	.	.
*Chromium	65/ 166	18.20	UG/L	1.82E-02	0.00	1.00E+00	./	.	.	.
Benzoic Acid	1/ 31	24.30	UG/L	6.07E-03	0.00	4.00E+00	./	.	.	.
Diethylphthalate	8/ 77	4.60	UG/L	5.75E-03	0.00	8.00E-01	./	.	.	.
Xylene, Total	1/ 80	6.49	UG/L	3.24E-03	0.00	2.00E+00	./	.	.	.

* Contaminant of Concern

Table 5.3. (continued)

PARAMETER	EFPC Proportion Detected	Mean EFPC Result	Measurement Units	EFPC Toxicity Score	EFPC % of Total Toxicity	Toxicity Factor	Background Proportion Detected	Mean BKGD Result	BKGD Toxicity Score	Radiation Measurement Uncertainty
Carcinogenic Score										
Benzo(a)pyrene	1/ 77	5.02	UG/L	3.66E-02	53.04	7.30E+00	./	.	.	.
*Arsenic	42/ 162	7.20	UG/L	1.26E-02	18.24	1.75E+00	./	.	.	.
*Beryllium	18/ 166	1.27	UG/L	5.46E-03	7.91	4.30E+00	./	.	.	.
Benzo(a)anthracene	1/ 77	5.03	UG/L	3.67E-03	5.32	7.30E-01	./	.	.	.
Benzo(b)fluoranthene	1/ 77	5.03	UG/L	3.67E-03	5.32	7.30E-01	./	.	.	.
Benzo(k)fluoranthene	1/ 77	5.02	UG/L	3.66E-03	5.30	7.30E-01	./	.	.	.
Pentachlorophenol	1/ 68	17.40	UG/L	2.09E-03	3.03	1.20E-01	./	.	.	.
*Methylene Chloride	9/ 80	69.80	UG/L	5.24E-04	0.76	7.50E-03	./	.	.	.
Chrysene	1/ 77	5.03	UG/L	3.67E-04	0.53	7.30E-02	./	.	.	.
Heptachlor Epoxide	1/ 80	0.03	UG/L	2.34E-04	0.34	9.10E+00	./	.	.	.
bis(2-Ethylhexyl)phthalate	11/ 77	5.08	UG/L	7.11E-05	0.10	1.40E-02	./	.	.	.
Chloroform	2/ 80	5.76	UG/L	3.51E-05	0.05	6.10E-03	./	.	.	.
gamma-BHC (Lindane)	1/ 80	0.03	UG/L	3.34E-05	0.05	1.30E+00	./	.	.	.
4,4'-DDT	1/ 80	0.05	UG/L	1.75E-05	0.03	3.40E-01		.	.	.
RadionuclideScore										
Neptunium-237	8/ 79	11.40	PCI/L	2.51E-09	58.03	2.20E-10	./	.	.	10.7000
Americium-241	1/ 83	3.93	PCI/L	9.43E-10	21.76	2.40E-10	./	.	.	12.1000
*Total Radium	67/ 78	3.22	PCI/L	3.22E-10	7.44	1.00E-10	./	.	.	0.1290
*Uranium-238	60/ 60	10.20	PCI/L	2.86E-10	6.61	2.80E-11	./	.	.	0.1080
*Uranium-234	55/ 55	12.60	PCI/L	2.02E-10	4.66	1.60E-11	./	.	.	0.1250
Cesium-137	2/ 82	1.05	PCI/L	2.95E-11	0.68	2.80E-11	./	.	.	4.7600
Cobalt-60	3/ 82	1.27	PCI/L	1.90E-11	0.44	1.50E-11	./	.	.	3.4700
Uranium-235	58/ 65	0.55	PCI/L	8.78E-12	0.20	1.60E-11	./	.	.	0.0252
Strontium-89	24/ 78	2.65	PCI/L	7.95E-12	0.18	3.00E-12	./	.	.	0.6030
Strontium-90	3/ 58	-0.04	PCI/L	0.00E+00	0.00	3.60E-11	./	.	.	0.2020
EPA Risk Factor = None Available										
4-Nitrophenol	1/ 68	17.40	UG/L	.	.	.	./	.	.	.
Aluminum	78/ 166	10300.00	UG/L	.	.	.	./	.	.	.
Ammonia	32/ 71	0.58	MG/L	.	.	.	./	.	.	.
Cobalt	57/ 166	11.00	UG/L	.	.	.	./	.	.	.
Endosulfan II	1/ 80	0.05	UG/L	.	.	.	./	.	.	.
Fluoride	53/ 73	0.19	MG/L	.	.	.	./	.	.	.
Lead	66/ 164	13.90	UG/L	.	.	.	./	.	.	.
Nitrate/Nitrite	48/ 73	18.70	MG/L	.	.	.	./	.	.	.
Sulfide	27/ 52	3.03	MG/L	.	.	.	./	.	.	.
alpha-Chlordane	4/ 80	0.14	UG/L	.	.	.	./	.	.	.
p,p'-Methoxychlor	1/ 80	0.26	UG/L	.	.	.	./	.	.	.

* Contaminant of Concern

EPA Risk Factors were taken from the IRIS and HEAST Tables as updated through February, 1993.
Results less than detection were set to 1/2 DL (except for radionuclides).

compared to background. Cesium-137 was not measured at significant levels and will not be evaluated.

Hazard Score - Noncarcinogenic Effects

Metals that primarily contribute to the groundwater noncarcinogenic hazard score are antimony, arsenic, manganese, mercury, and thallium. Thallium was eliminated because of the low frequency of detection. With the exception of acetone (which was included as a COC), the other organic compounds in Table 5.3 were not considered because together they comprise less than 1% of the total risk.

Hazard Score - Carcinogenic Effects

Arsenic and beryllium are considered COCs and account for 18 and 8% of the total toxicity for carcinogens, respectively. Since PAHs were detected in only 1 out of 77 samples, their impact to groundwater will not be evaluated. PCBs were not detected in the groundwater samples. Pentachlorophenol was eliminated because it was infrequently detected. The remaining organics represent less than 1% of the total risk score from carcinogens.

Hazard Score - Radionuclides

Uranium -234, -235, and -238, and radium were measured in the groundwater samples. Since uranium-235 represented only 0.2% of the total risk score, it was eliminated as a COC. Since cesium-137, americium-241, and cobalt-60 counts were not significantly different from the background site and their measurement uncertainty was relatively high, they were not considered further. The strontium isotopes constitute less than 1% of the total risk from radionuclides, so they were not included as COCs.

Chemicals with no Score

Two metals were detected but could not be scored, since toxicity measures were not available. Aluminum and cobalt were detected in EFPC at concentrations that were not statistically different from the background site (t-test on mean values).

5.1.2.4 Baseflow surface water contaminants of concern

Metals:

barium, chromium, manganese, mercury, and zinc

Semivolatiles and volatiles:

acetone, bis(2-ethylhexyl)phthalate, chloroform, and tetrachloroethene

Pesticides:
aldrin, 4,4'-DDT, dieldrin, endrin, gamma-BHC, and heptachlor epoxide

Radionuclides:
uranium-234 and uranium-238.

Most of the chemical contaminants in EFPC surface water are found adsorbed to sediment or bound to suspended particulate matter. Under baseflow conditions (Table 5.4), fewer contaminants are detected (i.e., in dissolved form). Several metals and organic compounds were detected in EFPC waters, but at levels considerably below existing federal MCLs under SDWA. Observed levels were found to comply with the stringent federal ambient water quality criteria for the protection of human health and freshwater organisms. The measured activities of uranium-234 and uranium-238 are evaluated in the BRA. Neptunium-237, cesium-137, and cobalt-60 activities were low relative to their measurement uncertainty, so they are not considered further. Several organic compounds (pesticides, VOCs, and SVOCs) also were detected in the baseflow samples and have been included as COCs.

5.1.2.5 Disturbed water contaminants of concern

The EFPC risk assessment team collected "disturbed" water samples to simulate water column concentrations associated with recreational use of EFPC (e.g., swimming, wading). See Sect. 3 for details of sampling methods.

Metals:
barium, chromium, copper, manganese, mercury, and vanadium

Semivolatiles and pesticides:
bis(2-ethylhexyl)phthalate and dieldrin.

Hazard Score - Noncarcinogenic Effects

The metals that are responsible for the large majority (87%) of the toxicity score are manganese and mercury (Table 5.5). Two organic compounds, bis(2-ethylhexyl)phthalate and dieldrin, also were included as COCs based on the results of the concentration-toxicity screen.

Hazard Score - Carcinogenic Effects

Arsenic was detected only in the background samples, and therefore, was eliminated from consideration. Dieldrin and bis(2-ethylhexyl)phthalate were included as COCs based on the results of the concentration-toxicity screen.

Table 5.4 East Fork Poplar Creek Phase Ia and Ib results: toxicity scores based on average contaminant concentrations in EFPC base flow ranked by toxicity

PARAMETER	EFPC Proportion Detected	Mean EFPC Result	Measurement Units	EFPC Toxicity Score	EFPC % of Total Toxicity	Toxicity Factor	Background Proportion Detected	Mean BKGD Result	BKGD Toxicity Score	Radiation Measurement Uncertainty
----------	----------	----------	----------	Noncarcinogenic Score	----------	----------	----------	----------	----------	----------
*Manganese	13/ 14	24.300	UG/L	4.86E+00	44.40	5.00E-03	3/ 3	16.000	3.20E+00	.
*Heptachlor epoxide	2/ 11	0.021	UG/L	1.64E+00	14.97	1.30E-05	0/ 1	0.025	1.92E+00	.
*Aldrin	1/ 11	0.023	UG/L	7.69E-01	7.03	3.00E-05	0/ 1	0.025	8.33E-01	.
*Mercury	4/ 14	0.215	UG/L	7.17E-01	6.55	3.00E-04	0/ 3	0.100	3.33E-01	.
*Dieldrin	4/ 11	0.035	UG/L	6.99E-01	6.39	5.00E-05	0/ 1	0.050	1.00E+00	.
*Barium	14/ 14	41.300	UG/L	5.89E-01	5.39	7.00E-02	3/ 3	42.800	6.11E-01	.
*Tetrachloroethene	1/ 13	4.310	UG/L	4.31E-01	3.94	1.00E-02	0/ 3	3.330	3.33E-01	.
*Chloroform	1/ 13	4.190	UG/L	4.19E-01	3.83	1.00E-02	0/ 3	3.330	3.33E-01	.
*bis(2-Ethylhexyl)phthalate	1/ 13	4.810	UG/L	2.40E-01	2.20	2.00E-02	1/ 3	4.330	2.17E-01	.
*Endrin	1/ 11	0.046	UG/L	1.54E-01	1.41	3.00E-04	0/ 1	0.050	1.67E-01	.
*Zinc	12/ 14	25.800	UG/L	8.58E-02	0.79	3.00E-01	1/ 3	12.300	4.09E-02	.
Copper	0/ 14	3.080	UG/L	8.31E-02	0.76	3.70E-02	1/ 3	2.530	6.85E-02	.
*4,4'-DDT	3/ 11	0.039	UG/L	7.86E-02	0.72	5.00E-04	0/ 1	0.050	1.00E-01	.
*Acetone	3/ 13	6.310	UG/L	6.31E-02	0.58	1.00E-01	0/ 3	5.000	5.00E-02	.
*gamma-BHC (Lindane)	4/ 11	0.018	UG/L	5.85E-02	0.54	3.00E-04	0/ 1	0.025	8.33E-02	.
1,1,1-Trichloroethane	0/ 13	4.420	UG/L	4.91E-02	0.45	9.00E-02	0/ 3	3.330	3.70E-02	.
Diethylphthalate	2/ 13	4.380	UG/L	5.48E-03	0.05	8.00E-01	0/ 3	5.000	6.25E-03	.

* Contaminant of Concern

Table 5.4. (continued)

PARAMETER	EFPC Proportion Detected	Mean EFPC Result	Measurement Units	EFPC Toxicity Score	EFPC % of Total Toxicity	Toxicity Factor	Background Proportion Detected	Mean BKGD Result	BKGD Toxicity Score	Radiation Measurement Uncertainty
Carcinogenic Score										
*Dieldrin	4/ 11	0.035	UG/L	5.59E-04	43.55	1.60E+01	0/ 1	0.050	8.00E-04	.
*Aldrin	1/ 11	0.023	UG/L	3.92E-04	30.54	1.70E+01	0/ 1	0.025	4.25E-04	.
*Heptachlor epoxide	2/ 11	0.021	UG/L	1.94E-04	15.08	9.10E+00	0/ 1	0.025	2.28E-04	.
*bis(2-Ethylhexyl)phthalate	1/ 13	4.810	UG/L	6.73E-05	5.24	1.40E-02	1/ 3	4.330	6.07E-05	.
*Chloroform	1/ 13	4.190	UG/L	2.56E-05	1.99	6.10E-03	0/ 3	3.330	2.03E-05	.
*gamma-BHC (Lindane)	4/ 11	0.018	UG/L	2.28E-05	1.78	1.30E+00	0/ 1	0.025	3.25E-05	.
*4,4'-DDT	3/ 11	0.039	UG/L	1.34E-05	1.04	3.40E-01	0/ 1	0.050	1.70E-05	.
4,4'-DDD	2/ 11	0.042	UG/L	9.95E-06	0.77	2.40E-01	0/ 1	0.050	1.20E-05	.
Radionuclide Score										
Neptunium-237	0/ 13	5.150	PCI/L	1.13E-09	80.53	2.20E-10	0/ 3	0.863	1.90E-10	25.6000
*Uranium-238	12/ 12	4.130	PCI/L	1.16E-10	8.21	2.80E-11	./ .	.	.	0.1120
Cesium-137	0/ 14	2.730	PCI/L	7.65E-11	5.43	2.80E-11	0/ 3	2.760	7.73E-11	11.8000
*Uranium-234	11/ 11	3.680	PCI/L	5.88E-11	4.18	1.60E-11	./ .	.	.	0.1070
Cobalt-60	1/ 14	1.320	PCI/L	1.98E-11	1.41	1.50E-11	0/ 3	2.000	3.01E-11	8.3200
Uranium-235	12/ 12	0.216	PCI/L	3.45E-12	0.25	1.60E-11	./ .	.	.	0.0258

* Contaminant of Concern

Table 5.4. (continued)

PARAMETER	EFPC Proportion Detected	Mean EFPC Result	Measurement Units	EFPC Toxicity Score	EFPC % of Total Toxicity	Toxicity Factor	Background Proportion Detected	Mean BKGD Result	BKGD Toxicity Score	Radiation Measurement Uncertainty
---EPA Risk Factor = None Available---										
Aluminum	3/ 14	173.000	UG/L	.	.	.	0/ 3	68.200	.	.
BOD	8/ 8	5.080	MG/L	.	.	.	1/ 1	5.000	.	.
DISSOLVED ORTHO-PO4	10/ 10	0.937	MG/L	.	.	.	1/ 1	0.013	.	.
Dissolved Organic Carbon	10/ 10	2.110	MG/L	.	.	.	1/ 1	1.900	.	.
Endosulfan I	1/ 11	0.023	UG/L	.	.	.	0/ 1	0.025	.	.
Endosulfan II	1/ 11	0.046	UG/L	.	.	.	0/ 1	0.050	.	.
Endosulfan sulfate	5/ 11	0.033	UG/L	.	.	.	0/ 1	0.050	.	.
Nitrate/Nitrite	10/ 10	3.970	MG/L	.	.	.	1/ 1	0.500	.	.
Total Alkalinity	10/ 10	136.000	MG/L	.	.	.	1/ 1	201.000	.	.
Total Organic Carbon	10/ 10	8.640	MG/L	.	.	.	1/ 1	8.000	.	.
Total Suspended Solids	10/ 10	0.960	MG/L	.	.	.	1/ 1	10.800	.	.
Trichloroethene	1/ 13	4.120	UG/L	.	.	.	0/ 3	3.330	.	.

EPA Risk Factors were taken from the IRIS and HEAST Tables as updated through February, 1993.
Results less than detection were set to 1/2 DL (except for radionuclides).

Table 5.5. East Fork Poplar Creek Phase Ia and Ib results: toxicity scores based on average contaminant concentrations in EFPC disturbed water ranked by toxicity

PARAMETER	EFPC Proportion Detected	Mean EFPC Result	Measurement Units	EFPC Toxicity Score	EFPC % of Total Toxicity	Toxicity Factor	Background Proportion Detected	Mean BKGD Result	BKGD Toxicity Score	Radiation Measurement Uncertainty
Noncarcinogenic Score										
*Manganese	4/ 4	269.00	UG/L	5.38E+01	74.43	5.00E-03	1/ 1	643.00	1.29E+02	.
*Mercury	4/ 4	2.68	UG/L	8.92E+00	12.35	3.00E-04	0/ 1	0.10	3.33E-01	.
Arsenic	0/ 4	1.50	UG/L	5.00E+00	6.92	3.00E-04	1/ 1	5.20	1.73E+01	.
*Vanadium	2/ 4	8.40	UG/L	1.20E+00	1.66	7.00E-03	1/ 1	7.20	1.03E+00	.
*Barium	4/ 4	79.20	UG/L	1.13E+00	1.57	7.00E-02	1/ 1	121.00	1.73E+00	.
*Dieldrin	1/ 4	0.04	UG/L	7.75E-01	1.07	5.00E-05	0/ 1	0.05	1.00E+00	.
*Copper	4/ 4	18.50	UG/L	5.00E-01	0.69	3.70E-02	1/ 1	13.40	3.62E-01	.
*bis(2-Ethylhexyl)Phthalate	3/ 4	9.50	UG/L	4.75E-01	0.66	2.00E-02	1/ 1	10.00	5.00E-01	.
Zinc	4/ 4	68.70	UG/L	2.29E-01	0.32	3.00E-01	1/ 1	31.10	1.04E-01	.
Endrin	1/ 4	0.04	UG/L	1.29E-01	0.18	3.00E-04	0/ 1	0.05	1.67E-01	.
Fluoranthene	1/ 4	4.00	UG/L	1.00E-01	0.14	4.00E-02	0/ 1	6.00	1.50E-01	.
*Chromium	3/ 4	7.80	UG/L	7.80E-03	0.01	1.00E+00	1/ 1	7.10	7.10E-03	.
Carcinogenic Score										
Arsenic	0/ 4	1.50	UG/L	2.63E-03	77.72	1.75E+00	1/ 1	5.20	9.10E-03	.
*Dieldrin	1/ 4	0.04	UG/L	6.20E-04	18.34	1.60E+01	0/ 1	0.05	8.00E-04	.
*bis(2-Ethylhexyl)Phthalate	3/ 4	9.50	UG/L	1.33E-04	3.94	1.40E-02	1/ 1	10.00	1.40E-04	.
EPA Risk Factor = None Available										
Aluminum	4/ 4	5690.00	UG/L	.	.	.	1/ 1	4690.00	.	.
Cobalt	2/ 4	5.48	UG/L	.	.	.	1/ 1	8.60	.	.

* Contaminant of Concern

EPA Risk Factors were taken from the IRIS and HEAST Tables as updated through February, 1993.
Results less than detection were set to 1/2 DL (except for radionuclides).

Chemicals with no Score

Two metals were detected but could not be scored, since no toxicity measures were available. Aluminum and cobalt were detected in EFPC at concentrations that were not statistically different from the background site (t-test on mean values).

5.1.3 Determination of Exposure Point Concentrations

Once the environmental monitoring data have been validated, they must be manipulated for the purposes of risk assessment. The data must be aggregated and statistical calculations must be performed to derive a meaningful estimate of the exposure point concentrations.

5.1.3.1 Data aggregation

The area under investigation in the BRA is quite large and characterized by a number of different land use types and habitats. In order for risk assessment to be meaningful, the analysis must focus on key receptors at potential risk of exposure and the land use area and circumstances under which exposure is most likely to occur. The concept of the "exposure unit" may be introduced as a basis for the assessment of exposure. An exposure unit may be thought of as the geographic area within which a receptor would realistically be expected to spatially and temporally aggregate exposure to contaminants.

In a residential area, the most appropriate exposure unit may be an area the size of a backyard (i.e., a quarter of an acre). In an agricultural setting, the exposure unit may be larger, encompassing perhaps an area of an acre or more. The important point is that the exposure unit or area under evaluation sets the boundaries or basis for aggregating data for the purpose of deriving exposure point concentrations. The discussion that follows presents the logic behind data aggregation and statistical evaluation for each environmental medium under investigation.

In this risk assessment, EFPC (including the associated floodplain) and the SLB have been considered as separate units in the RI. Therefore, data aggregation was performed separately for these two areas.

EFPC and Floodplain

Within EFPC and the floodplain, data were aggregated using different methodologies. For surface water, groundwater, and sediments, all data were grouped together in a single set. For floodplain soils, three different approaches were used, with each approach offering a finer resolution of the location and level of contamination. These three approaches involve aggregating data according to segments, aggregating data according to land use within each segment, and kriging.

Surface Water, Groundwater, and Sediments. All validated data for EFPC surface water (from the length of the creek) were aggregated and treated as a single data set. The data were then statistically manipulated to derive the mean and the 95% upper confidence limit (UCL) on the arithmetic mean (see Sect. 5.1.4). Similarly, validated data from groundwater sampling and sampling of sediments were each treated as single large data sets for risk assessment purposes. The reason for adopting this convention is explained more fully in the exposure assessment section of the BRA. In essence, however, it is based on the assumption that residents of EFPC are free to wander the creek and may be exposed to surface water and sediments at any location along the system.

With regard to groundwater, no one is currently using groundwater as a source of drinking water in the vicinity of EFPC and is unlikely to do so in the future. Further, local areas or distinct zones of elevated groundwater concentrations have not been identified. Therefore, the full groundwater data set has been used to derive the exposure point concentrations for residents at any location along EFPC. The data for each of these media were compiled and summarized statistically. The summary tables are presented at the end of Appendix L.

Floodplain Soils. A different approach has been used for data aggregation of floodplain soils. As discussed in the following sections, risk assessment has been conducted as a function of land use area. Ideally, if time and cost were not constraints, the monitoring program could generate a data set of sufficient spatial density that exposure units could be defined within each land use area, and would serve as the basis for data aggregation. The EFPC monitoring program was comprehensive and achieved wide spatial coverage. However, even with the thousands of samples collected, the data set is inadequate to meaningfully support the derivation of exposure point concentrations for small exposure units within each land use area. Therefore, three approaches were adopted to achieve a higher level of spatial resolution from the existing monitoring data.

First, the EFPC floodplain was divided along the length of the creek into nine segments (see discussion in Sect. 2). Maps 2 and 3 (Vol. 5) depict the segmentation of EFPC for the purposes of the BRA. These segments were established based on an understanding of the nature and extent of contamination, and a knowledge of current and projected future land uses. Each of these segments was effectively treated as a homogeneous unit with regard to data aggregation and estimation of exposure point concentration. In this manner, all data within a given segment were combined to calculate a single exposure point concentration, which would represent the level of contamination encountered anywhere in the segment. A discussion of the rationale for classification of each floodplain segment land use type and pertinent physical and cultural features within each segment is provided in Sect. 2.2.1. Because mercury is the most widely distributed

contaminant in EFPC and accounted for more than 75% of the total toxicity in the scoring/selection of COCs, the monitoring data for mercury weighs more heavily in segmentation decisions than other contaminants. The exposure point concentrations developed from aggregating data according to this approach are then used in the risk equations to calculate carcinogenic and noncarcinogenic risk (see Sects. 5.2 and 5.4).

The second approach was to aggregate the monitoring data within each segment according to land use. Note that more than one land use is often defined within each segment. For example, Segment 1 currently contains both commercial and open land uses, and also could include residential land use in the future [see Maps 2 and 3 (Vol. 5)]. Data from each land use area were aggregated so that exposure point concentrations could be calculated separately for each land use area within each segment. The land use area exposure point concentrations were then compared to the segment-wide exposure point concentrations (described in the previous paragraph). The results of this comparison are discussed in Sect. 5.2.3.3.

The third approach involves using geostatistical interpolation methods for spatial evaluation of contaminant concentrations (i.e., kriging). Kriging is a weighted moving average interpolation method that uses the best linear unbiased estimator by weighing the adjacent sample values to calculate an average value for a given region or block. Due to the large geographic areas under evaluation and the unavoidable limitations of the data set, this additional approach was employed in an effort to partially overcome the limitations of the sampling program.

The assistance of Dr. Evan Englund at EPA-Environmental Monitoring Systems Laboratory in Las Vegas (EMSL/LV) and Dr. A.K. Singh at the Environmental Research Center, University of Nevada-Las Vegas was obtained to perform the kriging analysis of the Phase Ib soil transect NAA sample data (see Sect. 3.2.4). Dr. Singh evaluated the creek in sections to accommodate the computer software limitations and to account for variations between different sections of the creek.

A set of maps (see Map 10) was generated for the upper soil layer (0 to 41 cm), with average mercury concentrations within blocks (of 20 $\times$ 20 meter dimensions) overlain on the floodplain. These maps are color coded for 50 and 200 parts per million (ppm) of mercury. These concentration codings were selected because of the preliminary remediation goals calculated for contaminated soil (Sect. 7) of 58 ppm for children and 198 ppm for adults based on combined soil ingestion and dermal contact.

The data generated from the kriging analysis offer a finer resolution of the mercury concentrations in floodplain soils. In the BRA of EFPC, these data may be used in two principal

ways: in subsequent analysis of baseline risks to human health (RI), and as a basis for comparison with remediation goals (cleanup levels) and more accurately delineating areas of the creek for which remediation is required (FS). At this time, the data from EPA-EMSL/LV were not used in the BRA.

The goal of these three approaches for the floodplain soils is to provide successively higher degrees of resolution. For creekwide aggregation, a single upper-bound exposure point concentration is calculated to represent the contamination level for the whole creek (e.g., as for sediment, groundwater, and surface water samples). When the data are aggregated according to segment, nine exposure point concentrations are calculated (corresponding to the nine segments). When the data are aggregated according to land use within each segment, multiple exposure point concentrations are calculated within each segment (corresponding to the number of land uses within that segment). This allows a finer resolution of the areas and levels of contamination. Kriging, a form of data aggregation, would allow the risk assessor to specify an even smaller area (e.g., an area the size of a backyard), and predict location and levels of contamination within that smaller area.

Sewer Line Beltway (SLB)

For the SLB, a single land use classification has been assumed for data aggregation purposes. Over its entire length, the SLB has areas that can be classified as residential, commercial, or open land use types. However, only open land use is applicable to those areas that were sampled with the highest levels of contamination (see Sect. 3.2.4 and Map 9). All of these areas are immediately adjacent to a primary street and are not used for any specific purpose other than as a buffer to residential or commercial properties or for occasional pedestrian traffic. Three contaminated areas will be evaluated individually: a small area at the intersection of Tulane Avenue and the Oak Ridge Turnpike, Fairbanks Road from Emory Valley Road to Warehouse Road, and Emory Valley Road west from the intersection with Fairbanks Road (see Map 9).

5.1.3.2 Statistics

Once the analytical data from the laboratory have undergone quality assurance/quality control (QA/QC) evaluation and COCs have been selected, summary statistics are prepared for each chemical and environmental medium using the validated data. These summary tables are presented at the end of Appendixes L, M, and P and provide the following information:

- sampling event, location, and period;
- detection limits;
- frequency of detection;

- the minimum, maximum, and mean concentrations of chemicals in the environmental medium;
- the reasonable maximum exposure (RME) concentration (i.e., the 95% UCL on the arithmetic mean); and
- background concentrations.

"Not detected" results for chemical contaminants have been treated as one-half the limit of detection and included in the calculation of the arithmetic means (note that a different convention is adopted for radionuclides). Sampling results characterized by the "J" qualifier for organic compounds (i.e., the chemical has been identified and a concentration is estimated, but the observed level is below the contract required quantitation limit) or the "B" qualifier for inorganics (i.e., less than the contract required detection limit, but greater than the instrument detection limit) have been included in the data set and the calculation of the mean. Results for analysis of blanks have been compared with field samples. Field samples were considered "positive" values and included in the data set only if these results passed the "5× or 10×" rule specified by EPA for interpretation of blank data (EPA 1989a).

5.1.3.3 Calculation of the exposure point concentration

EPA recommends use of the arithmetic mean in deriving a representative and conservative exposure point estimate (EPA 1989a; 1992c,d). The Agency specifies that the average concentration is most appropriately used because:

- the derivation of carcinogenic and chronic noncarcinogenic toxicity criteria is based on lifetime average exposures, and
- the average concentration is most representative of the concentration to which a receptor at a given site would be exposed over time.

EPA specifies the use of the average concentration regardless of the shape of the underlying distribution of the environmental quality data (EPA 1992c,d). The Agency notes that the geometric mean of a set of sampling results bears no logical relationship to the cumulative intake that would result from long-term (i.e., chronic) exposure to site-related contaminants. The geometric mean is considered an inappropriate basis for estimating the concentration term in Superfund exposure assessment.

EPA now requires two types of exposure estimates for Superfund risk assessments. Given the uncertainty associated with any point estimate of the true average exposure concentration at a site, EPA recommends use of the 95% UCL on the arithmetic mean concentration (EPA 1989a; 1992c,d). An RME concentration (i.e., the UCL) and an arithmetic mean concentration should

both be used. EPA defines the RME estimate as "the highest exposure that could reasonably be expected to occur for a given exposure pathway at a site" (EPA 1992c). In the present study of EFPC, both the arithmetic mean and the UCL have been used in characterizing exposure point concentrations.

EPA recommends two methods for deriving the RME (or UCL) exposure point concentration. As discussed in the recent supplement to Risk Assessment Guidance for Superfund (RAGS) (EPA 1992c), EPA indicates that choice of the appropriate method depends upon a knowledge of the underlying distribution of the sampling data from the exposure area of concern. Although EPA recommends use of the arithmetic mean as the average exposure point concentration regardless of the form of the underlying distribution, the Agency specifies the need to test the distribution of the data set before deriving the UCL.

If the underlying data are lognormally distributed, EPA indicates that the UCL of the arithmetic mean should be determined as follows:

$$UCL = e^{\left(\bar{x} + 0.5s^2 + \frac{sH}{\sqrt{n-1}}\right)} \quad (1)$$

where

UCL	=	95th percentile UCL on the arithmetic mean,
e	=	exponential (natural log) equal to 2.718,
$\bar{x}$	=	the mean of the log transformed data,
s	=	the standard deviation of the log transformed data,
H	=	H statistic (from table) for computing a one-sided upper 95th percentile confidence limit on a lognormal mean (Gilbert 1987), and
n	=	sample size.

If goodness of fit tests indicate the data set is consistent with a normal distribution, EPA recommends use of the Student's t statistic in deriving the UCL on the arithmetic mean. As follows:

$$UCL = \bar{x} + t_{1-\alpha,\, n-1} \cdot \left(\frac{s}{\sqrt{n}}\right) \quad (2)$$

where

t	=	student t-statistic (Gilbert 1987), and
α	=	upper-tail area.

In the BRA of EFPC, equation (2) has been used to estimate the UCL on the arithmetic mean exposure point concentration. The EFPC risk assessment team believes that this method should be used regardless of the underlying shape of the distribution of the data set. A receptor exposed to contaminants from a given study area will integrate exposure both spatially and temporally. For example, consider a 30-year exposure period over which an adult comes into contact with contaminated soil at a given site. For the first year, the individual wanders the site and may be said to experience an averaged (i.e., mean) site concentration over this time. The same situation occurs with regard to each of the remaining 29 years of exposure where the receptor is integrating contact with site contamination and experiencing an averaged exposure point concentration. Following the central limit theorem, the distribution of mean values (i.e., the integrated exposure point concentrations experienced by a receptor over time) is always normally distributed regardless of the underlying sampled distributions. Therefore equation (2) above may be appropriately used.

The above discussion notwithstanding, for small, highly skewed data sets, the issue of normality verses lognormality becomes more important. For larger data sets (i.e., $n > 50$) with few outliers, selection between the methods becomes less critical. It is important to recognize that if a data set is characterized by considerable variability and the sample size is small, the UCL may exceed the maximum observed value in the data set. In this case, EPA recommends use of the maximum observed concentration as the RME (UCL) estimate (EPA 1989a). In the evaluation of EFPC, the UCL is established as the value derived using equation (2) or the maximum observed value, whichever is smaller. The data used in the human health BRA are summarized by creek segment and are presented in Appendixes L and M.

5.1.4 Uncertainty in the Analytical Data

Uncertainty will always surround estimates of environmental concentrations at hazardous waste sites. The objective is to understand, minimize, and quantify this uncertainty while evaluating chemical results. The risk assessor must consider several issues when establishing the exposure point concentration: sampling program design, analytical methods, and data aggregation methods.

Procedures were established during sampling and analysis design to reduce uncertainty surrounding surface water and groundwater results. To reduce the influence of high and low water events (i.e., flooding and drought conditions), groundwater and surface water samples were collected at various times, locations, and under different conditions. Replicate samples also were collected. The results were compared and contrasted over time between sampling events to establish trends or concentration patterns (see Appendix E).

Sediments are transportable and sensitive to flooding events. The approach taken when sampling sediments during the EFPC RI was to rely on results from composite sediment samples collected from three sites spaced 200 meters apart to characterize the contaminant load for each 600-meter reach of EFPC. This approach was used to model the transient nature of the sediments.

A larger degree of uncertainty is associated with soil and sediment sample results than aqueous sample results based on a single set of samples for each location. Although soil is relatively immobile, analytical results are only a representation of the aliquot that was actually measured. The concept of support (EPA 1989a) was defined to minimize the sample variance throughout the floodplain. Three characteristics (i.e., three-dimensional volume, shape, and orientation) taken together are referred to as support and were used to identify the sampling units. Samples were collected for the EFPC RI to represent a particular depth integrated concentration of either 12 (during Phase Ia) or 16 in. (during Phase Ib).

The heterogeneity of the soil matrix also increases uncertainty associated with soil and sediment sample results. As observed from the EFPC pilot study (Sect. 3.2.4), wide ranges of concentrations were identified due to the heterogeneity of the soil matrix. This was confirmed by the vertical integration study, which showed stratification of mercury and other contaminants by specific horizons. The study was conducted to ascertain the form of mercury in the floodplain soils (Appendix G) using a scanning electron microscope. The study concluded that mercury, uranium, and thorium were present in the soils as discrete particles in varying sizes and disbursement patterns. Consequently, metal contaminants are not uniformly present throughout the soil medium and characterization with the same confidence as surface water or groundwater is more difficult.

The NAA/cold vapor atomic absorption (CVAA) equivalency study (Table I.11, Appendix I) distinguished the contribution of the individual components to variability among sample results (i.e., differences between laboratories, analytical methods, sample locations, sample stations at the same location, and samples collected from the same station). The report concluded that 16 to 22% of the total was due to variability within the sample itself. If the variability due to the difference in sample stations, which is the nature of characterizing an area within the floodplain, was added to the variability of a single sample, the total was raised from 46 to 62%. In conclusion, results can be considered valid (i.e., accurate and precise) for an individual sample location, but the degree of uncertainty increases dramatically when extending a characterization any distance from that location.

5.2 EXPOSURE ASSESSMENT

The objective of the BRA is to evaluate the current and future risks to human health and to determine the need for site remediation. Exposure assessment is a critical component of this process. It is here that contact, or the potential for contact, with contamination originating from the study area is measured or quantified. Exposure assessment is used to examine the movement or transport of chemicals through the environment, from source of release to point of exposure, and to estimate the amount of contaminant available at a biological boundary (i.e., the skin, lungs, intestinal tract, or other target organ or tissue). In the BRA for EFPC, exposure assessment is conducted to:

- identify the receptors at potential risk of contact with contaminants,
- determine the exposure pathways of importance, and
- quantify intake or dose for all contaminants and pathways of concern.

The focus of the exposure assessment for EFPC is principally the determination of chronic or long-term intake or dose. ORNL CRAC has requested that a subchronic risk assessment be conducted as part of the preliminary screening-level assessment to help identify exposure pathways of concern. This assessment has been conducted and summarized in the BRA. However, the chronic exposure estimates are of primary importance in evaluating longer-term current and future exposure to contaminants in EFPC. It is these estimates that will be principally used in determining the need for site remediation.

5.2.1 Characterization of Exposure Setting

In order to identify exposure pathways and quantify intake or dose, it is necessary to acquire an understanding of the general characteristics of the study area, the land uses, and the receptors potentially at risk of exposure.

5.2.1.1 Overview

EFPC and its tributaries are the major drainage system for the DOE Y-12 Plant and the city of Oak Ridge, Tennessee. The floodplain of EFPC begins inside the Y-12 Plant (i.e., at Lake Reality) and travels west through the city of Oak Ridge to a point just below the K-25 Site where it joins with Poplar Creek. Although the site history and environmental setting have been described in detail in Sects. 2 and 3, this information has been summarized briefly to help focus the exposure assessment. EFPC surface water, sediments, and floodplain soils have been principally affected by contaminant discharge from the Y-12 Plant for more than three decades.

The repository of contamination in these environmental media is the focus of the BRA and this exposure assessment.

EFPC became contaminated as a result of lithium processing operations at the Y-12 Plant. Mercury, used in the production of enriched lithium, was released to the environment along with smaller quantities of other inorganic and organic contaminants. Although the lithium process was shut down in 1963, the discharge of small quantities of mercury continues to this day. Mobilization of residual contamination in sumps, sewers, and cavities beneath the Y-12 Plant following storm flow events is the principal source of this continuing release.

The EFPC watershed is a rectangular area approximately 9 miles long and 3.5 miles wide. The creek itself is approximately 14 miles long. The drainage area is characterized by a diversity of land use types. Lower EFPC flows downstream from Lake Reality at the Y-12 Plant through engineered channels; through the commercial and residential areas of Oak Ridge; passing through more rural, privately owned properties; and finally onto the DOE Oak Ridge Reservation to its confluence with Poplar Creek. The creek has been channelized as it passes through some residential areas of Oak Ridge. Drainage ditches and culverts that cross the floodplain discharge into EFPC at many locations. The creek depth varies widely throughout the length of the system and may range from only several inches deep to more than 6 feet at a number of locations.

The human health exposure assessment for EFPC begins with an analysis of current and projected future land use. Land use maps have been developed to support this effort. These maps, in Vol. 5, present the current land use for the EFPC floodplain, the region of the SLB, and the city of Oak Ridge. Based on an understanding of zoning, existing land use, and constraints and incentives to development, a second map of projected or future land use also has been created.

Four land use classifications are depicted on Maps 2 and 3 in Vol. 5: agricultural, residential, commercial, and open land use. These land use types provide the basis for development of exposure scenarios used in the human health risk assessment:

- exposure in the agricultural setting or "homesteader" scenario: current and future,
- exposure to residential populations: current and future,
- exposure in the commercial (nonindustrial) setting: current and future, and
- exposure associated with occasional use of open land: current and future.

Each exposure scenario is defined by a set of exposure pathways. These pathways are discussed in detail in the following sections.

Although EFPC is posted by the city of Oak Ridge, the creek is accessible for recreational activities such as swimming and wading. Consideration of these activities has been incorporated into the agricultural, residential, and open land use scenarios. It is reasoned that a separate recreational scenario is appropriate only for those individuals who do not reside in the community surrounding EFPC and who occasionally use the creek for recreational activities. These individuals are projected to be exposed much less frequently than the population that permanently lives and works in the area. The open land use scenario is essentially a recreational use assessment for nonresidents and includes hiking and trespassing as well as swimming and wading.

The exposure assessment as described facilitates derivation of separate risk estimates (i.e., for receptors at risk of exposure) as a function of the exposure scenario/land use type and the observed levels of contamination in the defined land use area under investigation. The results of sampling and analysis for the EFPC system will be segregated geographically. Monitoring data for a given area along the creek will be used in deriving exposure point concentrations for use in risk assessment.

The receptor groups at greatest risk of exposure are assumed to be children and adults who reside in the community along EFPC. For each exposure scenario and receptor group, the intensity, duration, and frequency of exposure will be characterized. Due to the difficulty associated with realistically quantifying human exposure (i.e., measures of intake or dose), the exposure evaluations will be based on a series of estimates or evaluations. Risk assessment will be conducted using RME values (i.e., an upper-bound conservative estimate) and most likely exposure (MLE) values (i.e., an average or representative value). The RME estimate is most commonly used by EPA and is discussed in RAGS Vol. 1, Part A (EPA 1989a).

In the evaluation of EFPC, RME and MLE estimates will be developed for environmental concentrations, as well as for all input variables used in the exposure assessment equations, and used to estimate chronic intake or dose. This convention is recommended by several of the EPA regional offices and has been adopted in this assessment as a first effort to bound the risk estimates and serve as a preliminary basis for characterizing uncertainty. ORNL CRAC has recommended use of a conservative and nonconservative screening-level assessment to identify exposure pathways of concern. The conservative and nonconservative estimates correspond to the RME and MLE evaluations of this study.

In addition to the use of RME and MLE estimates, the risk assessment will be conducted using probability density functions (PDFs) and Monte Carlo simulation to generate distributions of risk estimates. PDFs will be used instead of point estimates for key input variables in the

exposure and risk characterization equations. More information is provided on this approach in Sects. 5.2.3 and 5.5.

The RME estimates frequently serve as a basis for risk management and define the highest exposure that is reasonably expected to occur at a site. EPA Region IV has requested use of the RME estimate as the principal basis for risk estimates in this study. RME estimates make selective use of upper-bound (90th or 95th percentile) values for exposure variables. Because they are based on a number of conservative (health-protective) assumptions, RME values will tend to overestimate the actual exposure to chemicals in a given environmental medium for a given exposure pathway. However, this is useful in providing an upper-bound estimate of exposure for a particular scenario and will ensure that projected risks do not underestimate the true risks likely to occur at the site. MLE estimates, on the other hand, are based on average values for exposure variables.

The RME estimate of intake or dose for a given exposure pathway (i.e., pathway-specific RME) should *not* be based solely on upper-bound or RME estimates for all input variables. EPA recommends selecting values for intake variables such that the combination of these values represents a 90th percentile estimate of intake (EPA 1989a,e). An example of this would be to combine the upper 95% confidence limit of the arithmetic mean contaminant concentration with 90th percentile values for contact rate, and 50th percentile values for exposure frequency, duration, and body weight. In establishing exposure assumptions for an RME, it is important that the evaluation not become a worst-case scenario.

Similarly, the RME estimates for combined exposure across pathways (i.e., scenario-specific RMEs) should not be based solely on RME estimates for each component exposure pathway. EPA guidance specifies (EPA 1989a):

> "Only if you can explain why the key RME assumptions for more than one pathway apply to the same individual or subpopulation should the RME risks for more than one pathway be combined. . . It may be [more] appropriate to combine one pathway's RME risks with other pathways' risk estimates that have been derived from more typical exposure parameter values."

This EPA guidance will be considered in the human health risk assessment for EFPC. In general, the use of RME and MLE assumptions along with a probabilistic characterization of uncertainty will provide a meaningful perspective of the range of exposures that may be experienced by individuals at risk of exposure.

5.2.1.2 Exposure scenarios

As discussed above, the four exposure scenarios under evaluation in the baseline human health risk assessment are:

- exposure to residential populations: present and future,
- exposure in the agricultural setting or homesteader scenario: present and future,
- exposure in the commercial (nonindustrial) setting: present and future, and
- exposure in an open land use setting to nonresidents: present and future.

Figure 5.1 presents a conceptual site model for the BRA depicting the transport and exposure pathways of concern. Table 5.6 provides an overview of the specific exposure assessments to be conducted as part of the risk assessment and presents the four exposure scenarios under evaluation, the exposure pathways that define the exposure scenarios, and an indication whether current and future exposures are considered. Each of the exposure scenarios is discussed below, and the defining exposure pathways are discussed in detail in Sect. 5.2.2. The rationale for inclusion or removal of each pathway from consideration is addressed in Sect. 5.2.3.1.

Residential Exposure Scenario. Residential receptors are defined as those individuals (residents of Oak Ridge, Tennessee) who live and work in proximity to EFPC. These individuals are assumed to make recreational use of floodplain soils, swim or wade in the creek, raise a small garden, consume homegrown produce, and occasionally ingest fish obtained from EFPC. Residents of this area are not currently using groundwater as a source of potable water. However, there are no constraints to the use of groundwater, and this will be considered a possible future exposure pathway.

The relevant current and future exposure pathways for the residential scenario are:

- dermal exposure to surface water while swimming,
- dermal exposure to surface water while wading,
- incidental ingestion of surface water while swimming,
- dermal exposure to sediments while wading,
- dermal exposure to soil,
- incidental ingestion of soil,
- ingestion of homegrown produce, and
- ingestion of recreationally caught fish.

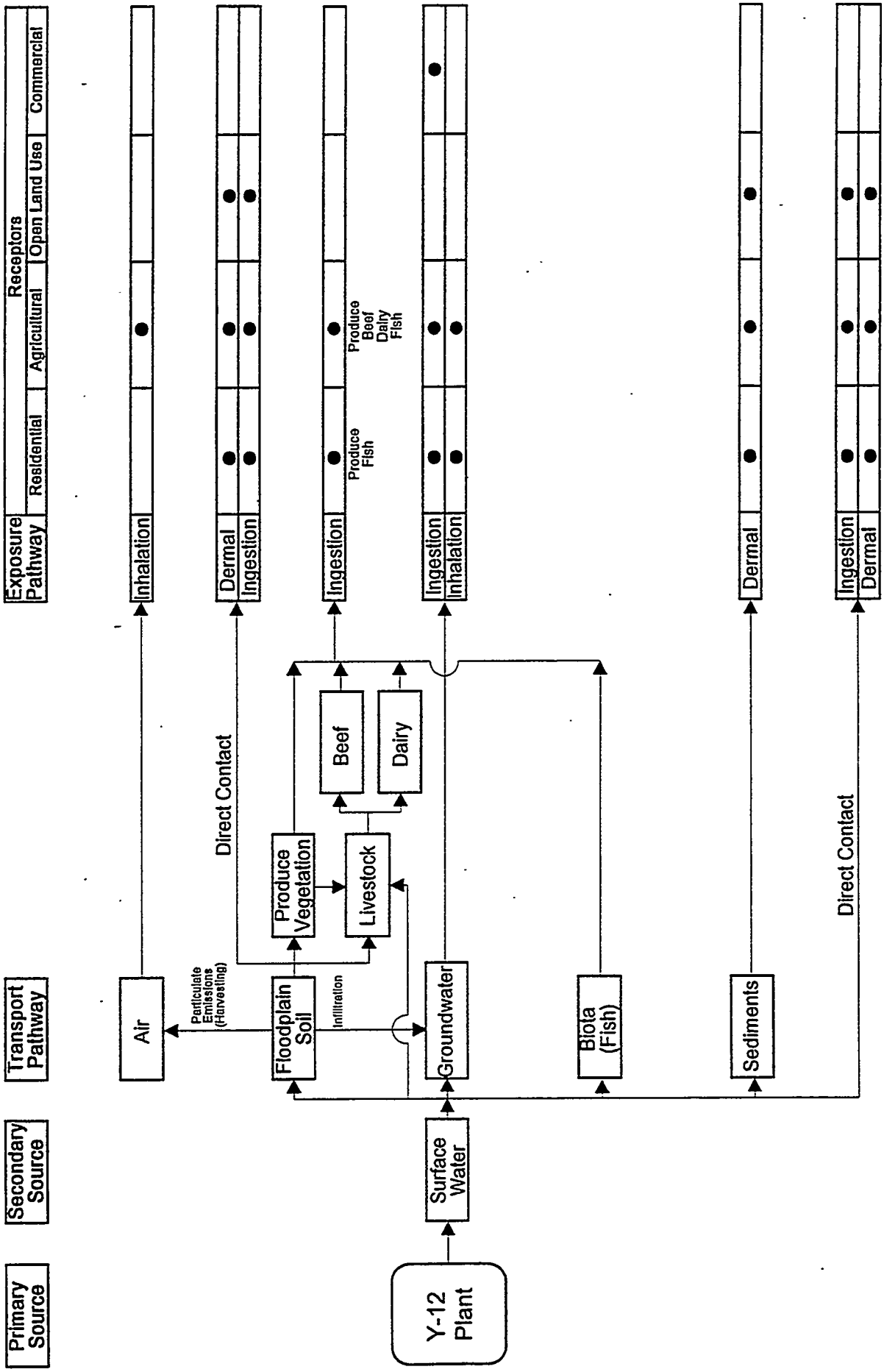

Fig. 5.1. Conceptual site model: human health risk assessment

Table 5.6. Scenarios and associated pathways used in the human health exposure assessment of EFPC

PATHWAY	SCENARIO							
	Residential		Agricultural		Open Land Use		Commercial	
	Present	Future	Present	Future	Present	Future	Present	Future
Surface Water								
Dermal while swimming	●	●	●	●	●	●		
Dermal while wading	●	●	●	●	●	●		
Inadvertent ingestion	●	●	●	●	●	●		
Sediment								
Dermal contact with sediment	●	●	●	●	●	●		
Groundwater								
Ingestion		●		●				●
Inhalation of vapors (showering)		●		●				
Soil								
Dermal	●	●	●	●	●	●		
Ingestion	●	●	●	●	●	●		
Air								
Mowing (particulates)			●	●				
Food								
Homegrown/locally grown:								
• fruits and vegetables	●	●	●	●				
• meat			●	●				
• milk/dairy				●				
Recreationally caught:								
• fish	●	●	●	●				

Note: Residential and agricultural scenarios include recreational activities.

Values for parameters used to evaluate each pathway may not be the same for all scenarios. The parameter values under each scenario are shown in Tables 5.7 and 5.8.

In addition to these pathways, evaluation of future exposure potential also will include exposure to groundwater through ingestion and inhalation (vapor phase chemicals) routes, as shown in Table 5.6.

In this scenario, risks to both adults and children are assessed. The age groups evaluated for children are from 3 to 12 years for the RME and 6 to 9 years for the MLE. The RME range was chosen because it was thought that children aged 3 to 12 years would cover the range of ages that would spend the most time in the contaminated floodplain area. Children younger than 3 years would more likely play in or immediately around the home and would generally be supervised by an adult. Children older than 12 years would be more likely to spend time at other locations outside the residential setting. Ages 6 to 9 (the middle portion of the RME range) were chosen for the MLE using best professional judgment.

Agricultural/Homesteader Exposure Scenario. The agricultural/homesteader scenario evaluates exposure to farm families living and working in the EFPC floodplain. Receptors may be exposed to contaminants present in or released from EFPC via a number of pathways. Exposure pathways include all of those common to the residential scenario described above, including recreational elements, such as swimming and wading exposure to surface water and sediments, ingestion exposure to recreationally caught fish, and future exposure to groundwater. Elements of the exposure assessment that are unique to the agricultural/homesteader scenario include ingestion of meat and milk/dairy products obtained from cattle raised on land on EFPC, and inhalation exposure to particulates released during harvesting of hay. The frequency of exposure to produce (i.e., fruits and vegetables) also is projected to be greater for the farm family.

As designed, there are no substantial differences in exposure assumptions for many of the exposure pathways for the residential and homesteader scenarios. This was considered appropriate given the current very limited agricultural activity along EFPC. At two locations, beef cattle and horses are raised in the vicinity of floodplain soils. No farms in the immediate area raise chickens or dairy cattle, or crops on a commercial scale. Based on the available information on activities along EFPC, it has been assumed that residents in the agricultural scenario maintain small home gardens of the same nature as families living in a residential setting.

For those pathways common to the agricultural/homesteader and residential scenarios, values for the exposure variables are the same as those presented for the equivalent residential exposure pathways (Sect. 5.2.2). The following pathways are included in the agricultural/homesteader scenario:

- dermal exposure to surface water while swimming,
- dermal exposure to surface water while wading,

- incidental ingestion of surface water while swimming,
- dermal exposure to sediments while wading,
- dermal exposure to soil,
- incidental ingestion of soil,
- ingestion of local or homegrown produce,
- ingestion of recreationally caught fish,
- ingestion of home-raised beef,
- ingestion of home-produced milk (future exposure only),
- exposure to groundwater (future exposure only) via ingestion and inhalation of vapors, and
- inhalation of particulates while mowing.

As with the residential exposure scenarios, the assessment evaluates both adults and children (RME: 3 to 12 years; MLE: 6 to 9 years).

Commercial Receptor Exposure Scenario. Commercial property along EFPC is located primarily on the eastern portion of the system. With the exception of the DOE facilities, no large industrial plants are located along EFPC. The commercial property consists primarily of small businesses and strip shopping areas. Although contiguous to the floodplain, none of the commercial properties is located in the floodplain. Commercial zones are built upon paved areas a distance from the creek. Given this situation, individuals working in the commercial areas are not at risk of direct exposure to contaminants in floodplain soils.

The exposure assessment assumes there is no exposure of commercial receptors to contaminants in EFPC under the current land use scenario. Given the moist floodplain soils and vegetated cover, inhalation exposure to entrained particulates is not a concern. Exposure under the commercial land use scenario is limited to a hypothetical future use of groundwater by the business community. The only pathway that is evaluated is ingestion of groundwater by adult receptors.

In actuality, this is an overly conservative assumption, given that municipal water is currently available, and groundwater is not a source of drinking water and is unlikely to be a source in the future. However, in an attempt to prepare a thorough assessment and to conservatively bound the potential risks to human health, the groundwater pathway is included.

Open Land Use/Trespasser Exposure Scenario. The open land use/trespasser scenario is designed to account for occasional exposure to EFPC soils, surface water, and sediments by individuals who are not residents of the EFPC area (i.e., their exposures are not captured under

either the residential or homesteader scenarios). This scenario evaluates exposure to children and adults. Exposure to children under this scenario focuses on ages 9 to 18 for the RME. Children aged 12 to 15 years are used in evaluating the MLE, as it is more likely that hiking/trespassing will occur for this age group than for younger children.

The open land use scenario includes the following exposure pathways:

- dermal exposure to surface water while swimming,
- dermal exposure to surface water while wading,
- incidental ingestion of surface water while swimming,
- dermal exposure to sediments while wading,
- dermal exposure to soil, and
- ingestion of soil.

The exposure variables used to calculate intake or dose for each of the pathways are similar to those used in the residential or agricultural/homesteader scenario. However, it has been assumed that exposure under the open land use scenario is characterized by shorter durations and frequencies than exposure for the agricultural/homesteader and residential scenarios (i.e., for individuals who live in the vicinity of EFPC).

5.2.1.3 Previous studies on exposure to EFPC soils

In 1985, a large-scale epidemiological study was conducted in Oak Ridge, Tennessee in an attempt to characterize actual exposure of residents to mercury found in the EFPC floodplain (Rowley et al. 1985). A primary objective of this study was to obtain human body levels of mercury from Oak Ridge residents known to have been exposed to contaminated soils. Mercury levels were measured in hair and urine samples in both exposed and unexposed populations. The study concluded that urine and hair mercury levels did not differ significantly between the exposed and unexposed populations, and that concentrations in both populations did not reach levels associated with known health risks.

5.2.2 Identification of Exposure Pathways

Detailed information on each of the exposure pathways for each identified land use scenario is presented in the discussion below and in Sects. 5.2.2.1 through 5.2.2.11. The general form of the intake on dose equation is presented below. For each exposure pathway, the intake or dose equations are presented along with the selected values for all exposure variables. Table 5.6 summarizes the land use scenarios and the exposure pathways they comprise. Table 5.7 comprehensively summarizes the selected RME and MLE values adopted for each of the exposure

Table 5.7. Summary of input variables for baseline public health risk assessment

Exposure Medium and Pathway	Residential				Agricultural/Homesteader				Open Land Use				Commercial			
	Child		Adult		Child		Adult		Child		Adult		Child		Adult	
	RME	MLE	RME	MLE	RME	MLE	RME	MLE	RME	MLE	RME	MLE	RME	MLE	RME	MLE
SURFACE WATER - CURRENT AND FUTURE EXPOSURE																
Swimming (Dermal)																
Skin Area (cm^2)	11173	9310	23000	20000	11173	9310	23000	20000	17333	14900	23000	20000				
Fraction Exposed	1	1	1	1	1	1	1	1	1	1	1	1				
Permeability Coefficient (cm/hr)	Chemical Specific				Chemical Specific				Chemical Specific							
Exposure Time (hr/d)	2.6	1	2.6	1	2.6	1	2.6	1	1	0.5	1	0.5				
Exposure Frequency (d/y)	16	7	16	7	16	7	16	7	5	1	5	1				
Exposure Duration (y)	9	3	30	9	9	3	30	9	9	3	30	9				
Body Weight (kg)	26	25	70	70	26	25	70	70	49	50.6	70	70				
Averaging Time (d)																
Carcinogens	25550	25550	25550	25550	25550	25550	25550	25550	25550	25550	25550	25550				
Noncarcinogens	3285	1095	10950	3285	3285	1095	10950	3285	3285	1095	10950	3285				
Wading (Dermal)																
Skin Area (cm^2)	11173	9310	23000	20000	11173	9310	23000	20000	17333	14900	23000	20000				
Fraction Exposed	0.38	0.21	0.38	0.21	0.38	0.21	0.38	0.21	0.38	0.21	0.38	0.21				
Permeability Coefficient (cm/hr)	Chemical Specific				Chemical Specific				Chemical Specific							
Exposure Time (hr/event)	2.6	1	2.6	1	2.6	1	2.6	1	1	0.5	1	0.5				
Events per day	1	1	1	1	1	1	1	1	1	1	1	1				
Exposure Frequency (d/y)	36	7	36	7	36	7	36	7	5	1	5	1				
Exposure Duration (y)	9	3	30	9	9	3	30	9	9	3	30	9				
Body Weight (kg)	26	25	70	70	26	25	70	70	49	50.6	70	70				
Averaging Time (d)																
Carcinogens	25550	25550	25550	25550	25550	25550	25550	25550	25550	25550	25550	25550				
Noncarcinogens	3285	1095	10950	3285	3285	1095	10950	3285	3285	1095	10950	3285				
Ingestion (while swimming)																
Ingestion Rate (L/hr)	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05	0.05				
Relative Absorption Factor	1	1	1	1	1	1	1	1	1	1	1	1				
Exposure Time (hr/d)	2.6	1	2.6	1	2.6	1	2.6	1	1	.5	1	.5				
Exposure Frequency (d/y)	16	7	16	7	16	7	16	7	5	1	5	1				
Exposure Duration (y)	9	3	30	9	9	3	30	9	9	3	30	9				
Body Weight (kg)	26	25	70	70	26	25	70	70	49	50.6	70	70				
Averaging Time (d)																
Carcinogens	25550	25550	25550	25550	25550	25550	25550	25550	25550	25550	25550	25550				
Non carcinogens	3285	1095	10950	3285	3285	1095	10950	3285	3285	1095	10950	3285				
SEDIMENT - CURRENT AND FUTURE EXPOSURE																
Wading (Dermal)																
Skin Area (cm^2)	11173	9310	23000	20000	11173	9310	23000	20000	17333	14900	23000	20000				
Fraction Exposed	0.38	0.10	0.38	0.10	0.38	0.10	0.38	0.10	0.38	0.10	0.38	0.10				
Adherence Factor (mg/cm^2)	0.5	0.3	0.5	0.3	0.5	0.3	0.5	0.3	0.5	0.3	0.5	0.3				
Dermal Absorption Factor	Chemical Specific				Chemical Specific				Chemical Specific							
Exposure Frequency (d/y)	36	7	36	7	36	7	36	7	5	1	5	1				
Exposure Duration (y)	9	3	30	9	9	3	30	9	9	3	30	9				
Body Weight (kg)	26	25	70	70	26	25	70	70	49	50.6	70	70				
Averaging Time (d)																
Carcinogens	25550	25550	25550	25550	25550	25550	25550	25550	25550	25550	25550	25550				
Noncarcinogens	3285	1095	10950	3285	3285	1095	10950	3285	3285	1095	10950	3285				

Table 5.7. (continued)

Exposure Medium and Pathway	Residential				Agricultural/Homesteader				Open Land Use				Commercial	
	Child		Adult		Child		Adult		Child		Adult		Adult	
	RME	MLE	RME	MLE	RME	MLE	RME	MLE	RME	MLE	RME	MLE	RME	MLE
GROUNDWATER - FUTURE EXPOSURE ONLY														
Ingestion														
Ingestion Rate (L/d)	0.88	0.52	2.00	1.40	0.88	0.52	2.00	1.40					1.00	0.7
Relative Absorption Factor	1	1	1	1	1	1	1	1					1	1
Exposure Frequency (d/y)	350	350	350	350	350	350	350	350					250	250
Exposure Duration (y)	9	3	30	9	9	3	30	9					25	9
Body Weight (kg)	26	25	70	70	26	25	70	70					70	70
Averaging Time (d)														
Carcinogens	25550	25550	25550	25550	25550	25550	25550	25550					25550	25550
Non carcinogens	3285	1095	10950	3285	3285	1095	10950	3285					9125	3285
Inhalation of Vapors														
Inhalation Rate (m^3/h)	0.6	0.6	0.6	0.6	0.6	0.6	0.6	0.6						
Volatilization Constant (L/m^3)	0.5	0.5	0.5	0.5	0.5	0.5	0.5	0.5						
Exposure Time (h/d)	0.42	0.25	0.25	0.12	0.42	0.25	0.25	0.12						
Exposure Frequency (d/y)	350	350	350	350	350	350	350	350						
Exposure Duration (y)	9	3	30	9	9	3	30	9						
Body Weight (kg)	26	25	70	70	26	25	70	70						
Averaging Time (d)														
Carcinogens	25550	25550	25550	25550	25550	25550	25550	25550						
Noncarcinogens	3285	1095	10950	3285	3285	1095	10950	3285						
SOIL - CURRENT AND FUTURE EXPOSURE														
Ingestion														
Ingestion Rate (mg/d)	133	50	100	40	133	50	100	40	133	50	100	40		
Relative Absorption Factor	1	1	1	1	1	1	1	1	1	1	1	1		
Exposure Frequency (d/y)	350	270	350	270	350	270	350	270	150	40	150	40		
Exposure Duration (y)	9	3	30	9	9	3	30	9	9	3	30	9		
Body Weight (kg)	26	25	70	70	26	25	70	70	49	50.6	70	70		
Averaging Time (d)														
Carcinogens	25550	25550	25550	25550	25550	25550	25550	25550	25550	25550	25550	25550		
Noncarcinogens	3285	1095	10950	3285	3285	1095	10950	3285	3285	1095	10950	3285		
Dermal Absorption														
Skin Area (cm^2)	11173	9310	23000	20000	11173	9310	23000	20000	17333	14900	23000	20000		
Fraction Exposed	0.47	0.10	0.47	0.10	0.47	0.10	0.47	0.10	0.47	0.10	0.47	0.10		
Adherence Factor (mg/cm^2)	1	0.6	1	0.6	1	0.6	1	0.6	1	0.6	1	0.6		
Dermal Absorption Factor	Chemical Specific				Chemical Specific				Chemical Specific					
Exposure Frequency (d/y)	350	270	350	270	350	270	350	270	150	40	150	40		
Exposure Duration (y)	9	3	30	9	9	3	30	9	9	3	30	9		
Body Weight (kg)	26	25	70	70	26	25	70	70	49	50.6	70	70		
Averaging Time (d)														
Carcinogens	25550	25550	25550	25550	25550	25550	25550	25550	25550	25550	25550	25550		
Noncarcinogens	3285	1095	10950	3285	3285	1095	10950	3285	3285	1095	10950	3285		

Table 5.7. (continued)

Exposure Medium and Pathway	Residential				Agricultural/Homesteader				Open Land Use				Commercial			
	Child		Adult		Child		Adult		Child		Adult		Child		Adult	
	RME	MLE	RME	MLE	RME	MLE	RME	MLE	RME	MLE	RME	MLE	RME	MLE	RME	MLE
FOOD - CURRENT AND FUTURE EXPOSURE																
Produce Ingestion																
Ingestion Rate (kg/d)[a]	0.262	0.090	0.340	0.113	0.262	0.090	0.340	0.113								
Fraction from Area (%)	0.35	0.23	0.35	0.23	0.35	0.23	0.35	0.23								
Relative Absorption Factor	1	1	1	1	1	1	1	1								
Exposure Frequency (d/y)	150	150	150	150	350	150	350	150								
Exposure Duration (y)	9	3	30	9	9	3	30	9								
Body Weight (kg)	26	25	70	70	26	25	70	70								
Averaging Time (d)																
Carcinogens	25550	25550	25550	25550	25550	25550	25550	25550								
Noncarcinogens	3285	1095	10950	3285	3285	1095	10950	3285								
Fish Ingestion																
Ingestion Rate (kg/d)[a]	0.042	0.014	0.054	0.023	0.042	0.014	0.054	0.023								
Fraction from Area (%)	0.05	0.01	0.05	0.01	0.05	0.01	0.05	0.01								
Relative Absorption Factor	1	1	1	1	1	1	1	1								
Exposure Frequency (d/y)	350	350	350	350	350	350	350	350								
Exposure Duration (y)	9	3	30	9	9	3	30	9								
Body Weight (kg)	26	25	70	70	26	25	70	70								
Averaging Time (d)																
Carcinogens	25550	25550	25550	25550	25550	25550	25550	25550								
Noncarcinogens	3285	1095	10950	3285	3285	1095	10950	3285								
Meat Ingestion																
Ingestion Rate (kg/d)[a]					0.074	0.030	0.100	0.040								
Fraction from Area (%)					0.75	0.44	0.75	0.44								
Relative Absorption Factor					1	1	1	1								
Exposure Frequency (d/y)					350	350	350	350								
Exposure Duration (y)					9	3	30	9								
Body Weight (kg)					26	25	70	70								
Averaging Time (d)																
Carcinogens					25550	25550	25550	25550								
Noncarcinogens					3285	1095	10950	3285								
FOOD - FUTURE EXPOSURE ONLY																
Milk/Dairy Ingestion																
Ingestion Rate (kg/d)[a]					0.614	0.256	0.400	0.160								
Fraction from Area (%)					0.75	0.44	0.75	0.44								
Relative Absorption Factor					1	1	1	1								
Exposure Frequency (d/y)					350	350	350	350								
Exposure Duration (y)					9	3	30	9								
Body Weight (kg)					26	25	70	70								
Averaging Time (d)																
Carcinogens					25550	25550	25550	25550								
Noncarcinogens					3285	1095	10950	3285								

[a] ingestion rates are wet weight.

Table 5.7. (continued)

Exposure Medium and Pathway	Residential				Agricultural/Homesteader				Open Land Use				Commercial			
	Child		Adult		Child		Adult		Child		Adult		Child		Adult	
	RME	MLE	RME	MLE	RME	MLE	RME	MLE	RME	MLE	RME	MLE	RME	MLE	RME	MLE
AIR - CURRENT AND FUTURE EXPOSURE																
Dust Inhalation (While Mowing)																
Inhalation Rate (m^3/h)							0.83	0.83								
Exposure Time (h/event)							8	8								
Exposure Frequency (events/y)							4	4								
Exposure Duration (y)							30	9								
Body Weight (kg)							70	70								
Averaging Time (d)																
Carcinogens							25550	25550								
Noncarcinogens							10950	3285								

factors for the residential, agricultural/homesteader, commercial, and open land use scenarios, and is formatted to facilitate comparison of values across scenarios, receptors, and exposure pathways. The derivation and references associated with each parameter are described in greater detail in Table M.1, Appendix M.

In Table 5.6, many pathways are shown to be common to more than one land use scenario. For example, all pathways in the residential land use scenario are also found in the agricultural/ homesteader scenario. Pathways common to the residential and agricultural scenarios are characterized (with few exceptions) by exposure variables of the same value (Table 5.7). Commonly, contact with contaminated environmental media is projected to be higher in the agricultural/homesteader setting than in the residential setting (e.g., greater inadvertent exposure to soils and higher home-grown produce ingestion rates). However, the evaluation of the EFPC agricultural/homesteader scenario is atypical in that no large-scale operating farms are located along the creek nor are any projected. The agricultural/homesteader scenario is differentiated from the residential scenario by inclusion of additional food chain pathways and an evaluation of exposure associated with harvesting of hay. Exposure factors for the commercial and open land use scenarios reflect circumstances specific to these pathways.

Ideally, exposure factors should be derived based on estimates of site-specific activities and behavior patterns of receptor groups at potential risk of exposure. Such information has not been compiled for EFPC and could only be meaningfully developed through a large-scale survey of residents of Oak Ridge, Tennessee. A survey of this magnitude is not believed to be warranted. In the absence of survey data, EPA guidance has been used whenever possible in selecting or deriving values for exposure variables. The principal sources of information used are the *Superfund Exposure Assessment Manual* (EPA 1988c), the *Exposure Factors Handbook* (EFH) (EPA 1989b), the *Risk Assessment Guidance for Superfund: Human Health Evaluation Manual* (RAGS) (EPA 1989a), the *Human Health Evaluation Manual, Supplemental Guidance: Standard Default Exposure Factors* (EPA 1991a), and the *Dermal Exposure Assessment: Principles and Applications* (EPA 1992b).

Table 5.7 summarizes the exposure factors for each of the pathways under evaluation. Pathways 1 through 13 present the following information:

- land use scenario,
- exposure variable name, and
- the RME and MLE estimates for each exposure variable.

In a deterministic assessment of risk to human health, it is common practice to assume that exposure factors (e.g., ingestion rates) remain relatively constant for adults over the duration of exposure. However, since exposure factors vary as children grow to adulthood, weighted-averaged values were used in characterizing the RME and MLE exposure to children over a specified time period.

For the residential and agricultural/homesteader scenarios, the RME exposure values for children are derived based upon averages over the exposure duration period of 9 years (ages 3 to 12). MLE values for children are time-weighted averages for the age group 6 to 9 years. In the commercial setting, RME and MLE values are based on exposure to adult receptors only. The RME exposure values here are based on a duration of 25 years, and the MLE assumes an exposure of 9 years. For the open land use scenario, the RME exposure values for children also are derived based on averages over the exposure duration period of 9 years (ages 9 to 18), while the MLE values for children are time weighted for the age group 12 to 15 years. Children of these ages were considered most likely to be involved in hiking or unsupervised use of open land along EFPC.

The exposure values are used to estimate chemical and radionuclide intake. Intake estimates for use in risk assessment are quantitative expressions of the amount of chemical (or radionuclide) available for subsequent absorption. Equations for intake are taken from RAGS (EPA 1989c).

Chemical intakes are estimated by means of the following general equation:

$$Intake\ mg/(kg \cdot d) = \frac{C \times IR \times EF \times ED \times CF}{BW \times AT} \qquad (3)$$

where

C = chemical concentration (exposure point concentration),
IR = intake rate,
EF = exposure frequency,
ED = exposure duration,
BW = average body weight,
AT = averaging time,
CF = dimensional conversion factors to attain appropriate units.

The above expression is the general form of the equation that will be used to derive estimates of subchronic or chronic intake or dose. Intake and dose estimates [in mg/(kg • d) and

pCi for radionuclides] will be developed for each substance of potential concern using the representative environmental concentrations (i.e., mean and UCL values). For radionuclides, intake estimates are based on the same general equation and those that follow, with the exception that there is no division by body weight or division by averaging time.

For lead, EPA has developed a computer program, LEAD 0.60 (EPA 1991e) that estimates blood lead uptake from various environmental sources. Blood lead levels are estimated in Sect. 5.4.4.4 and compared to a benchmark target blood lead level. LEAD 0.60 analyzes blood lead uptake in children, the most sensitive receptors of lead exposures, and is not currently applicable to adults. Therefore, analysis using LEAD 0.60 will be limited to children as potential receptors.

The uncertainty associated with the exposure variables is discussed in Sect. 5.2.3. A probabilistic risk assessment and a quantitative analysis of uncertainty are being conducted to supplement the more traditional EPA methods. In accomplishing this, probability distributions must be identified or derived for each exposure variable that will be treated stocastically. The information on these distributions makes explicit the variability or uncertainty surrounding the RME point estimates.

5.2.2.1 Dermal exposure to surface water while swimming

Contact with surface water is considered a recreational component of the residential, agricultural/homesteader, and open land use exposure scenarios. This pathway is not considered relevant for receptors in the commercial scenario. In addition to recreational use, surface water may be used for irrigation and hypothetically as a source of drinking water. The EFPC BRA does not incorporate surface water used for irrigation purposes. Results of surface water sampling indicated very low levels of contaminants under base flow conditions. Therefore, irrigation is considered a minor pathway, given that the assessment considers exposure to produce directly grown in floodplain soils. Use of surface water as a drinking water source is considered unlikely and of small consequence in any case given the low concentrations of site-related chemicals observed in dissolved form. However, as part of a conservative screening-level assessment, an evaluation has been conducted to document that hypothetical ingestion exposure to surface water is not a potential health problem.

For the residential and agricultural/homesteader scenarios, recreational exposure via the surface water pathway assumes that swimming occurs principally during the summer months (June through September), and that this activity is replaced by wading during the spring and autumn (April, May, and October). Dermal exposure to contaminated water is evaluated for both

children and adults who swim or wade in EFPC. The equation for calculating dermal exposure to contaminated water is:

$$Dose = C \times \frac{SA \times FE \times PC \times ET \times EF \times ED \times CF}{BW \times AT} \quad (4)$$

where

C = chemical concentration (μg/L),
CF = conversion factor (10^{-3}) (mg/μg) and (10^{-3} L/cm^3),
SA = total skin surface area available for contact (cm^2),
FE = fraction of skin surface area exposed (unitless),
PC = chemical-specific dermal permeability constant (cm/hr),
ET = exposure time (hr),
EF = exposure frequency (d/year),
ED = exposure duration (years),
BW = average body weight (kg),
AT = averaging time (days).

The exposure point concentration of chemicals in surface water includes dissolved chemicals and contaminants adsorbed onto fine sediments that may be resuspended during swimming or wading. Recreational activity in surface water (i.e., swimming and wading) increases the local concentration of suspended sediments. Surface water samples collected under base flow conditions may be used in developing exposure point concentrations. This may underestimate exposure during swimming if sediments are disturbed. Exposure to suspended sediments during recreational use of EFPC is estimated by using "disturbed" surface water samples. In addition to base flow samples, surface water samples were obtained that reflect (i.e., simulate) sediment disturbance and suspension likely to occur during swimming activities. Therefore, the exposure point concentration used in the risk assessment was the greater of the disturbed water or baseflow chemical concentration.

The skin surface area used to calculate exposure for all scenarios is based on the assumption of total body exposure during swimming. The values shown in Table 5.7 are means of the values for body surface areas for males and females of the appropriate age groups, as presented in the EFH (EPA 1989b). The swimming exposure pathway assumes that all of the skin will be exposed.

For the residential and agricultural scenarios, an exposure time of 2.6 hours per day is used to calculate RME exposure and an exposure time of 1 hour per day is used to calculate MLE exposure while swimming. In the absence of site-specific information, the recommended default value of 2.6 hours per day is used, which is the national average for swimming time (EPA 1989a); however, since this value may represent more than actual time in the water (EPA 1992b), a lower value is used for the MLE. In addition, swimming is assumed to occur 1 day per week during the summer months (June, July, August, and September) for the RME (i.e., 16 days per year), and 7 days per year for the MLE. The 7 day per year value is the national mean value for recreational swimming recommended by EPA (1988c; 1989a,b).

Under the open land use scenario, exposure time for swimming would be expected to be less than under the residential and agricultural scenarios. EPA (1992b) recommends an exposure time of 0.5 hour per event as a reasonable average value, and 1 hour per event for a reasonable upper value for a person who swims regularly. These recommendations are used for the MLE and RME exposure time values, respectively, for the open land use scenario. In the absence of site-specific information on swimming frequency, a value of 5 days per year is used to model the RME and 1 day per year is used to estimate the MLE under the open land use scenario.

Permeability coefficients (K_p) are chemical-specific measures of the potential for dermal transport. A limited number of these values are available in the literature. The published K_p values must be considered with care, since they may vary widely as a function of experimental design (i.e., as a function of variables such as vehicle, species, temperature, and concentration). A number of K_p values have been listed by EPA in the *Interim Guidance for Dermal Exposure Assessment* (EPA 1992b). Most of these values, however, have been derived based on structure-activity relationships. EPA (1992b) presents a steady-state and a nonsteady-state approach to estimating absorbed dose. For organic chemicals, EPA recommends use of K_p values predicted by correlation (by Potts and Guy) between log K_{ow} and molecular weight. For inorganics, EPA (1992b) recommends that a default K_p value of 10^{-3} cm/h be used.

Mean body weights used in the exposure assessment are derived from values for the different age groups presented in EPA (1989b). The values used in this assessment represent the combined mean of the male and female body weights for each age group. The 70-kg body weight value for adults is the value recommended by EPA (1989a,b).

Averaging time for evaluating noncarcinogenic effects is a pathway-specific value reflecting the actual chronic exposure period. As specified by EPA, averaging time values are determined by multiplying the exposure duration by 365 days per year. For carcinogens, the averaging time is based on a 70-year lifetime (i.e., 70 years $\times$ 365 d/year = 25,550 days).

5.2.2.2 Dermal exposure to surface water while wading

This pathway estimates exposure to residential and agricultural receptors, as well as occasional users of the creek. For all of these scenarios, dermal exposure through wading in EFPC is calculated in the same manner as dermal exposure through swimming [i.e., using equation (4) above]. However, different values are used for the fraction of surface area exposed and exposure frequency (Table 5.7). For all relevant exposure scenarios, the whole skin surface area is calculated the same as for the swimming pathway, and the fraction of the skin surface area exposed during wading is assumed to be limited to the extremities. For the RME, exposure is limited to the feet, legs, and hands, while for the MLE, exposure is assumed to occur on the feet, lower legs, and hands. The values for the surface area of these body parts were determined from the total body surface areas for adult males and females, and the percentage of the total surface area represented by each body part as presented in EPA (1989b, 1992b). Because percentages of total body surface area for body parts are not available for all of the ages within each of the 3-year age groups used in this assessment, the values were based on the assumption that the body parts in children represent the same fraction of the total as they do in adults.

For the residential and agricultural/homesteader scenarios, wading is assumed to occur during April, May, and October, when swimming is less likely because of lower temperatures. For the RME scenario, wading is assumed to occur 3 days per week during April, May, and October (i.e., 36 days per year), while for the MLE, it is assumed to be equal to the national average value for recreational swimming (i.e., 7 days per year). The values for exposure time are the same as those used in the scenario for dermal contact while swimming.

For the open land use scenario, wading is assumed to occur with the same exposure time and frequency as was used to estimate exposure while swimming. Therefore, the RME and MLE exposure times are 1 hour per event, and 0.5 hour per event, respectively, and the RME and MLE exposure frequencies are 5 days per year and 1 day per year, respectively. Chemical concentration, permeability coefficients, exposure duration, body weights, and averaging time values are as described for swimming.

5.2.2.3 Incidental ingestion of surface water while swimming

Incidental ingestion of surface water may occur during swimming. The equation for estimating intake of surface water via this pathway is:

$$Intake = C \times \frac{CR \times RAF \times ET \times EF \times ED}{BW \times AT} \qquad (5)$$

where

C = chemical concentration (mg/L),
CR = contact rate (L/hr),
RAF = relative absorption factor (unitless),
ET = exposure time (hr/d),
EF = exposure frequency (d/year),
ED = exposure duration (years),
BW = average body weight (kg),
AT = averaging time (days).

Ingestion exposure to surface water is characterized by the same frequency, duration, and time for swimming exposure for each respective exposure scenario. Chemical concentrations in the water are determined in a similar manner as for the swimming pathway. The default incidental ingestion rate during swimming is presented in EPA (1989a) as 0.05 L/h. This value is used for all age groups for both the RME and MLE assumptions. In addition, values for body weight and averaging times are the same as those cited for dermal exposure to surface water while swimming, for each respective exposure scenario.

The relative absorption factor (RAF) reflects the bioavailability following exposure to chemicals in environmental media, surface water in this case. This term is used to account for the differences in bioavailability of the contaminant in surface water (or any other environmental medium) and the contaminant in an experimentally administered medium (e.g., food) in laboratory test species (EPA 1989f). A RAF of 100% (i.e., RAF = 1) will be used in this assessment for all media.

5.2.2.4 Dermal exposure to sediments

Dermal exposure to sediments is projected to occur principally during recreational use of EFPC while children or adults wade in shallow waters. This pathway applies to receptors under the residential, agricultural/homesteader, and open land use scenarios. Incidental ingestion of sediments is not considered during wading activity. Ingestion of suspended sediment material has been incorporated into the evaluation of incidental ingestion of surface water while swimming. The equation for estimating dermal dose for sediments is:

$$Dose = C \times \frac{CF \times SA \times FE \times AF \times ABS \times EF \times ED}{BW \times AT} \qquad (6)$$

where

C = chemical concentration(mg/kg),
CF = conversion factor (10^{-6} kg/mg),
SA = total skin surface area available for contact (cm^2),
FE = fraction of skin surface area exposed (unitless),
AF = sediment to skin adherence factor (mg/cm^2),
ABS = dermal absorption factor (unitless),
EF = exposure frequency (d/year),
ED = exposure duration (years),
BW = average body weight (kg),
AT = averaging time (days).

The values used for total surface area and exposure frequency are the same as those used for dermal exposure to surface water while wading. As presented above, selection of fraction of skin surface area exposed during wading assumes that exposure is limited to the extremities. For the RME, exposure is limited to the legs, feet, and hands, while MLE exposure occurs on the feet and hands only. The values for the fraction of the total surface area of these body parts were determined as for the other dermal exposure pathways.

Estimates of direct dermal exposure to sediments require information on sediment adherence and percent absorption. (Note the distinction between direct sediment contact and contact with sediment material that has been suspended in the water column. This latter pathway is considered in the evaluation of dermal exposure to surface water.) Site-specific data are not available on adherence of EFPC sediments to human skin. EPA default values for sediment adherence are also not available. However, EPA indicates that adherence factors (AFs) for some sediments (particularly sandy sediments) are likely to be "much less" than for soils because contact with water may wash the sediments off the skin (EPA 1989a). Therefore, in the absence of data, AFs are set equal to one-half the value recommended by EPA for exposure.

In RAGS (EPA 1989a), EPA specified AF values for kaolin clay and potting soil of 2.77 and 1.45 mg/cm^2, respectively. Recent guidance from EPA Headquarters and Regions IV and X indicates that these values are now considered unrealistically high. EPA Headquarters and Region IV have specified a range of 0.2 to 1.0 mg/cm^2 as appropriate for use in evaluating dermal exposure to surface soil (EPA 1992b). EPA Region X, in their *Supplemental Risk Assessment Guidance for Superfund* (EPA 1991c), suggests values of 1.0 and 0.6 mg/cm^2 for RME and MLE (average) exposure estimates, respectively. The risk assessment uses one-half the AFs recommended by EPA Regions IV and X in the assessment of dermal exposure to

sediments during wading. Chemical concentrations are the upper-bound or average concentrations in surface sediments.

Percent absorption values for dermal exposure to sediment contaminants are chemical-specific. Values for this parameter may not be available for all of the COCs. EPA Region IV has issued interim guidance (default values) for absorption from the soil matrix (EPA 1992a). In the absence of chemical-specific values, the default values of 1.0% for dermal absorption of organics and 0.1% for dermal absorption of inorganics (i.e., metals) are used.

For the residential and agricultural/homesteader scenarios, wading is assumed to occur during April, May, and October, when swimming is less likely because of lower temperatures. For the RME scenario, exposure duration is assumed to be 3 days per week during April, May, and October (i.e., 36 days per year), while for the MLE, it is assumed to be equal to the national average value for recreational swimming (i.e., 7 days per year). For the open land use scenario, the exposure frequency is similar to that used for the wading surface water exposure pathway.

5.2.2.5 Groundwater ingestion: future exposure pathway

Residents of Oak Ridge do not currently use groundwater as a source of drinking water, and this pathway is currently unlikely to impact residents near EFPC. However, there are no constraints to the use of this resource as a potable water supply in the future. Therefore, ingestion of contaminated groundwater is included in the BRA as a future exposure pathway for residential, agricultural, and commercial receptors. This pathway is evaluated using the following equation:

$$Intake = C \times \frac{IR \times RAF \times EF \times ED}{BW \times AT} \qquad (7)$$

where

C = chemical concentration (mg/L),
IR = ingestion rate (L/d),
RAF = relative absorption factor (unitless),
EF = exposure frequency (d/year),
ED = exposure duration (years),
BW = average body weight (kg),
AT = averaging time (days).

For the residential and agricultural exposure scenarios, the RME is evaluated using the default adult drinking water ingestion rate of 2 liters per day for adults [90th percentile drinking water ingestion rate presented in EPA (1989a, 1991a)]. The MLE adult ingestion rate is the average value of 1.4 liters per day presented in RAGS. Ingestion rates for children are developed from intake values for tap water and total beverage intake by age group presented in EPA (1989b). The tap water is used for evaluating the MLE and the total beverage ingestion rates are used for the RME. The age groups that most closely match the groups in this assessment are used.

Exposure frequency for drinking water ingestion is 350 days per year. This value is based on daily ingestion from the contaminated source minus 2 weeks per year for vacation (EPA 1991a). Values for exposure duration, body weights, and averaging times are the same as described for previous pathways. Chemical concentrations are estimated through current monitoring.

For the commercial receptor, different values are adopted for ingestion rate, exposure frequency, and exposure duration for this exposure pathway. The recommended default drinking water ingestion rate for the commercial scenario is 1 liter per day. This value, recommended in EPA (1991a), is used for the RME estimate. The recommended ingestion rate of 1 liter per day is one-half of the RME drinking water ingestion rate of residential and agricultural/homesteader receptors. Therefore, the MLE ingestion rate for commercial receptors is 0.7 liters per day, which is one-half the MLE ingestion rate for residential and agricultural/homesteader receptors. Exposure frequency is limited to 250 days per year and duration is 25 years for the RME and 9 years for the MLE. The RME exposure duration is the default value presented in EPA (1991a). The MLE value is based on professional judgment.

5.2.2.6 Inhalation of vapors from groundwater: future exposure pathway

Future exposure to groundwater also may include the inhalation pathway. During bathing or showering, chemicals in vapor form or present in aerosol may be released to the ambient air. In EPA (1989a), the Agency recommends estimating exposure for this pathway using the following equation if sufficient site-specific data are available to project ambient air concentrations:

$$Intake = C_{vapor} \times \frac{IR \times ET \times EF \times ED}{BW \times AT} \quad (8)$$

where

C_{vapor} = estimated chemical concentration in the vapor (mg/m^3),
IR = inhalation rate (m^3/hr),
ET = exposure time (hr/d),
EF = exposure frequency (d/year),
ED = exposure duration (years),
BW = average body weight (kg),
AT = averaging time (days).

In the absence of critical site-specific information required to model air concentrations, the EPA Risk Assessment Forum recommends that the risk assessor assume that inhalation exposure to volatile chemicals during showering and bathing is equivalent to exposure from ingesting 2 liters of the same water per day (EPA 1988d). EPA Region I notes that although chemicals released from water into indoor air may present risks comparable to those associated with ingestion of groundwater, exposure models have not been extensively validated (EPA 1989f). EPA Region IX has indicated that they will accept the use of this generic model in risk assessments provided that intake/dose estimates are evaluated using EPA-verified inhalation reference concentrations (RfCs) (EPA 1989e).

EPA Region X has recommended a default approach for estimating indoor inhalation exposure to volatiles from contaminated water (EPA 1991c). Indoor air concentrations of volatile organics in household water are estimated assuming an upper-bound default volatilization constant of 0.5 L/m^3. This constant is based on experimental data from the work of Andelman (1990), who derived an equation defining the relationship between the concentration of contaminants in household water and the average concentration of the volatilized contaminants in air. EPA Headquarters also has adopted this approach and recommends its use in RAGS Vol. 1, Part B (EPA 1991c). This approach is recommended only for VOCs with Henry's constant > 10^{-5} atm • m^3/mole and a molecular weight less than 200 g/mol and has been adopted in the risk assessment of exposure to volatiles in groundwater at EFPC.

The default inhalation rate that is recommended in EPA (1989a) for evaluating this pathway is 0.6 m^3/h for all age groups. This value is used in conjunction with exposure times, frequencies, and durations presented for the showering/bathing pathway. The body weights and averaging times are the same as those used for the other exposure pathways.

Dermal exposure to contaminated groundwater is not evaluated as a future exposure pathway. Although dermal exposure occurs during showering and bathing, the magnitude of potential

exposures via dermal absorption is very low due to the short duration of skin contact. A limited number of chemicals have been observed in groundwater in the vicinity of EFPC, and these have generally been detected at low levels. Many of the chemicals found are inorganic compounds that exhibit low percutaneous absorption, particularly at dilute levels projected for hypothetical future groundwater use. For these reasons, EPA Region I commonly recommends that this exposure pathway not be quantitatively evaluated in the BRA (EPA 1989f).

5.2.2.7 Incidental ingestion of soil

Incidental ingestion of soil may occur in all age groups, and result from inadvertent hand to mouth contact. The exposure through this pathway is estimated using the following equation:

$$Intake = C \times \frac{IR \times RAF \times CF \times FI \times EF \times ED}{BW \times AT} \quad (9)$$

where

C = chemical concentration (mg/kg),
IR = ingestion rate (mg/d),
RAF = relative absorption factor (unitless),
CF = conversion factor (10^{-6} kg/mg),
FI = fraction ingested from a contaminated source (unitless),
EF = exposure frequency (d/year),
ED = exposure duration (years),
BW = average body weight (kg),
AT = averaging time (days).

Default values for soil ingestion rates are provided in EPA (1989a) and EPA's *Supplemental Guidance to RAGS* (EPA 1991c). EPA recommends the use of 200 mg/d for children under 6 years old, and 100 mg/d for all older ages (i.e., for RME estimates). The ingestion rates are 90th percentile values. EPA also recommends the use of these values in combination with body weights of 16 kg and 70 kg, respectively. To refine the estimates of intake, the evaluation of children potentially exposed to EFPC soils is based on time-weighted averages for the age groups under evaluation (i.e., time-weighted average intake values for ages 3 to 12 years, and for 6 to 9 years).

For the open land use scenario, soil ingestion is evaluated using the same basic methods as applied for the residential and agricultural/homesteader scenarios. While occasional users of the

creek will not be exposed for the whole day, at the recommendation of EPA Region IV, the full daily ingestion rate is used regardless of the number of hours per day the receptor is exposed. The age groups involved also differ from the residential scenario. The ingestion rate is 100 mg/d for all potential receptors, because all exposed individuals under this scenario are assumed to be more than 6 years old.

For residential and agricultural receptors, RME estimates of exposure frequency assume daily contact 350 days per year. The MLE estimates are based on the assumption of daily exposure for 9 months of the year (i.e., 270 days per year). This is based on the assumption that inclement weather or clothing prevents dermal exposure to the extremities for an equivalent of 3 months per year.

The range of exposure frequencies under the open land use scenario is substantially greater than that used to model the residential or agricultural/homesteader scenarios. An RME value of 150 days per year describes a person walking across the floodplain on his/her way to work or school, while an MLE value of 40 days per year describes a person who occasionally wanders on the contaminated area. The MLE value is based on the default central value for soil contact presented in EPA (1992b). The fraction of soil ingested from the contaminated source is assumed to be 100%.

5.2.2.8 Dermal exposure to soil

Exposure to soil through dermal contact is assumed to occur in adults and children as a result of gardening, house work, or recreational activities. The equation for estimating dermal dose is:

$$Dose = C \times \frac{CF \times SA \times FE \times AF \times ABS \times EF \times ED}{BW \times AT} \qquad (10)$$

where

C = chemical concentration (mg/kg),
CF = conversion factor (10^{-6} kg/mg),
SA = total skin surface area available for contact (cm^2),
FE = fraction of skin area exposed (unitless),
AF = soil to skin adherence factor (mg/cm^2),
ABS = absorption factor (unitless),
EF = exposure frequency (d/year),

ED = exposure duration (years),
BW = average body weight (kg),
AT = averaging time (days).

Ranges of values for the different parameters used to evaluate this exposure pathway are presented in Appendix M, Table M.1 (Pathway 8). The fraction of the skin surface area exposed in this pathway is derived assuming limited exposure to the extremities. For the RME, exposed extremities are assumed to be the legs, feet, upper arms, forearms, and hands. For the MLE, exposed extremities are limited to the feet and hands. Values for total surface area are determined in a similar manner as for the surface water wading pathway.

Estimates of dermal exposure to soils require information on soil adherence factors and percent absorption. Site-specific data are not available on adherence of EFPC floodplain soil to human skin. As noted above, EPA Region IV has suggested AF values of 0.2 to 1.0 mg/cm^2 (EPA 1992a) and Region X (EPA 1991c) has suggested AF values of 1.0 and 0.6 mg/cm^2 for RME and MLE (average) exposure estimates, respectively. The risk assessment uses the AFs recommended by Region IV in the assessment of dermal exposure to soils.

Percent absorption values are also chemical specific. Values for this parameter are not available for all of the COCs. EPA Region IV has issued interim guidance (default values) for absorption from the soil matrix (EPA 1992a). In the absence of chemical-specific values, the default values of 1.0% for dermal absorption of organics, and 0.1% for inorganics (i.e., metals) have been used. Dermal exposure may be substantially less than the 24 hours assumed in the development of dermal absorption factor values. However, the percent absorbed value cannot be scaled to an hourly rate, since this rate of absorption is not linear. Therefore, a conservative assumption is made that soils will remain attached to the skin for a whole day.

The values for exposure frequency for the residential, homesteader/agricultural, and open land use scenarios are the same as for the soil ingestion pathway for each respective scenario, since these two pathways are assumed to occur concurrently.

Chemical concentrations are the upper-bound or average values detected in soil. Exposure duration, body weights, and averaging times used for this pathway are the same as those described for the previous pathways.

5.2.2.9 Inhalation of particulates during harvesting

This exposure pathway is limited to homesteader/agricultural receptors. In this exposure pathway, it is assumed that the homesteaders cut hay that has been grown in the vicinity of floodplain soils. Mechanized equipment would likely be used, and contaminated respirable airborne particulates would be entrained in the ambient air during mowing (or cutting). Contaminants in the air would be present due to adherence to the cut vegetation and from disturbance of the surficial layer of soil.

Exposure assumptions have been selected to conservatively reflect exposure potential. It is assumed that the power mower operator would be at greatest risk of exposure due to proximity to the source of dust and would be the basis of the RME estimate. Based upon professional judgment and discussions with farm owners near EFPC, it is assumed that the RME and MLE receptor has a single field planted with hay and mows (harvests) this lot four times per year (8 hours per event).

Ambient air quality data are not available and were not collected for use in estimating atmospheric concentrations of suspended material associated with mowing activities. Since no direct measurements are available, contaminant concentrations in the respirable dust were estimated for mowing operations based on the methods developed by Eckerman and Young (1980). The following equation was used:

$$C_{air} = C_{soil} \times SD \times D \times RF \qquad (11)$$

where

C_{air} = estimated concentration of chemical in air (mg/m^3),
C_{soil} = measured concentration of chemical in soil (mg/kg),
SD = density of soil (kg/m^3),
D = soil depth from which resuspension may occur (m),
RF = resuspension factor.

The methods of Eckerman and Young (1980) and Sehmel (1984) make use of an empirically derived particulate resuspension factor. In the absence of site-specific data, ORNL CRAC has recommended use of a resuspension factor of 1×10^{-6}/m for mechanical harvesting of hay. The resuspension factor, in conjunction with soil density and soil concentration, yields an estimate of contaminant mass (adsorbed to airborne particulates) per cubic meter of air.

The soil density of the Newark soil series (found at the NOAA site, Bruner's Site, and Hinds Creek Transect A) ranged from 1.2×10^{-3} kg/m^3 to 1.4×10^{-3} kg/m^3, and the soil density of the Hamblin soil series (found at Sturm Site and Hinds Creek Transect B) ranged from 1.30×10^{-3} kg/m^3 to 1.45×10^{-3} kg/m^3. A soil density of 1.3×10^{-3} kg/m^3 was used to estimate the airborne concentrations of contaminants measured in the top 6 inches (= 0.01 m).

Once the concentration of suspended material in the ambient air (C_{Air}) is derived, the following equation is used to estimate intake for this pathway:

$$Intake = C_{air} \times \frac{IR \times ET \times EF \times ED}{BW \times AT} \quad (12)$$

where

C_{air} = estimated chemical concentration in the ambient atmosphere (mg/m^3),
IR = inhalation rate (m^3/hr),
ET = exposure time (hr/event),
EF = exposure frequency (events/year),
ED = exposure duration (years),
BW = average body weight (kg),
AT = averaging time (days).

No guidance is available from EPA regarding exposure during mowing/harvesting operations. The RME receptor (the mower operator) is assumed to be exposed over a 30-year working career (as an adult) and the MLE receptor is assumed to be exposed over a 9-year period. This pathway is considered relevant only for the mower operator (maximum exposure). No other receptors are included in this exposure pathway. The assumptions for the mowing pathway are presented in Appendix M, Table M.1 (Pathway 9).

Inhalation may be an exposure pathway of concern for chemicals that readily volatilize from soils or transport adsorbed on suspended particulates. Release of volatile chemicals to the atmosphere from soils in EFPC is not of concern due to the absence of VOCs in floodplain soils. Exposure to suspended particulates is considered insignificant given the moist floodplain soils. Neither of these pathways has been included in the BRA.

5.2.2.10 Food chain pathways

An essential component of the agricultural/homesteader scenario is the evaluation of food chain pathways. These pathways examine the potential exposure of human receptors to chemicals originating in floodplain soils that transport to (or concentrate in) vegetation, beef tissue, and dairy products. In the human health risk assessment for EFPC, estimates of exposure to contaminated produce, beef, and dairy are not based on measured concentrations. Concentrations in home-grown produce, beef, and dairy were estimated or "modeled" using bioconcentration and biotransfer factors (BCFs and BTFs, respectively) obtained from literature. Estimates are available for soil to plant BTFs, feed to beef tissue BTFs, and feed to milk BTFs (specifically designated B*v*, B*a*, and B*m*, respectively). In the absence of literature values, these transfer factors were derived using regression equations (for organic chemicals). In all cases, the use of the transfer factors assumes equilibrium partitioning between soil and biological tissue. Note that an experimental vegetation and garden study has been conducted in floodplain soils along EFPC. Data from this study, along with literature data, were used to supplement and verify the modeling of contamination in garden crops and are discussed in greater detail in the section entitled EFPC Vegetation and Garden Produce Study, and in Sect. 5.2.3.2.

Ingestion of Produce: Home Gardens

The produce ingestion pathway assumes that residential and agricultural receptors grow fruits and vegetables in backyard home gardens for their own consumption and that the gardens are grown in contaminated floodplain soils. However, only a small percentage of the total fruit and vegetable dietary intake of the receptors is projected to originate from this source. All food ingestion pathways are evaluated using the following equation:

$$Intake = Cp \frac{IR \times RAF \times FI \times EF \times ED}{BW \times AT} \qquad (13)$$

Should be WW

where

Cp = chemical concentration in produce (μg chemical/g produce ~~DW~~),
IR = ingestion rate of produce (g produce/d),
RAF = relative absorption factor (unitless),
FI = fraction ingested from contaminated area (unitless),
EF = exposure frequency (d/year),
ED = exposure duration (years),
BW = body weight (kg),
AT = averaging time (days).

In human health risk assessments, estimates of chemical concentration in produce typically are based on modeling of food chain transport through the use of published soil-to-plant transfer coefficients. This is the principal approach adopted in the BRA of EFPC. As a supplement to this approach, however, SAIC conducted an experimental study of contaminant uptake from EFPC floodplain soils in an attempt to empirically derive measurements of contaminant concentration in garden plant tissue. An overview of this effort is presented below in the subsection entitled EFPC Garden Produce and Vegetation Study.

Chemical concentrations in fruits and vegetables have been estimated primarily through the use of steady-state soil/vegetation transfer coefficients. The following equation is used:

$$Cp = Bv \times Sc \qquad (14)$$

where

Cp = chemical concentration in produce group (μg/g DW),
Bv = chemical and plant specific soil-plant biotransfer factor (μg chemical/g plant DW) per (μg chemical/g soil),
Sc = soil concentration of pollutant (μg chemical/g soil).

A number of surveys in scientific literature identify produce- and chemical-specific soil to plant biotransfer factors (Bvs) for inorganic contaminants. These include the EPA (1985c) series *Environmental Profiles and Hazard Indices for Constituents of Municipal Sludge*, Belcher and Travis (1989), Baes et al. (1984), Hoffman and Baes (1979), Ng et al. (1977), Ng (1982), NCRP (1991), NCRP (1989), and EPA (1990d). Table 5.8 compares Bv values identified from several of these key sources for COCs at EFPC, and shows the variability in the data.

The default Bv values developed in the EPA (1985c) series are chemical- and species-specific, and are based on peer reviewed experimental studies involving plant uptake of chemicals from soil. The EPA (1985c) values formed the basis Bvs for four product groups (i.e., leafy vegetables, root vegetables, garden fruit, and fresh legumes) presented by Belcher and Travis (1989) and EPA (1990d). Unfortunately, the data did not provide Bv values for all of the COCs at EFPC. Additional Bv values are presented by Ng et al. (1977) and Ng (1982). These Bv values are not produce-specific, but can be traced back to original field studies. This type of linkage to original peer-reviewed documentation is desirable, since it can be used to develop an understanding of the conditions under which uptake values were developed and the inherent limitations in the experimental methods that were used to derive such values.

Table 5.8. Soil to plant biotransfer factors

Chemical	NCRP-3 ((Ci/kg plant)/(Ci/kg soil))		NCRP-20 (Bq/kg plant)/(Bq/kg soil)		Baes et al. (conc. in plant DW/ conc. in soil DW)		Ng et al. (conc. in plant /conc. in dry soil)	
	Fresh Veg	Dry Forage	Fresh Veg	Dry Forage	Bv (veg portions)	Br (repro portions)	Bv (wet weight)	Cr (dry weight)
Aluminum			0.004	0.1	0.004	0.00065		
Arsenic			0.08	0.2	0.40	0.006		
Barium	0.01	0.1	0.01	0.1	0.15	0.015	0.0025	0.051
Beryllium			0.004	0.1	0.01	0.0015		
Cadmium			0.5	1	0.55	0.15		
Cobalt	0.03	0.4	0.08	2	0.2	0.007	0.03	0.24
Copper			0.05	0.8	0.4	0.25		
Cyanide								
Lead	0.01	0.09	0.1	0.5	0.45	0.009		
Manganese	0.4	3.0	0.3	10	0.25	0.05	0.36	2.6
Mercury	0.3	1.0	0.3	1	0.9	0.2		
Nickel	0.05	0.2	0.05	1	0.06	0.60	0.033	0.074
Silver	0.2	1.0	0.004	0.1	0.40	0.1		
Vanadium			0.002	0.1	0.0055	0.003		
Zinc	0.4	1.0	0.4	1	1.5	0.9	0.37	0.93
Americium-241	0.001	0.004	0.001	0.1	0.0055	0.00025		
Cesium 137	0.03	0.1	0.04	0.2	0.08	0.03	0.0095	0.089
Cobalt 60	0.03	0.4	0.08	2	0.02	0.007		
U-234, U-235, U-238	0.002	0.01	0.002	0.1	0.005	0.004		

Table 5.8. (continued)

Chemical	Ng et al.			Belcher and Travis (mg/kg plant DW)/mg/kg soil) (values are mean ± S.D.)				
	Bv (NRC) (WW)	Bv (other sources)(WW)	Cr (DW)	Leafy	Dry Leg	Fresh Leg	Root	Fruit
Aluminum								
Arsenic				0.62 ± 0.53	0.0006 ± 0.0003	0.0006 ± 0.0003	0.0102 ± 0.0148	0.93 ± 0.04
Barium	0.005	0.005						
Beryllium				0.006 ± 0.005	0.006 ± 0.005	0.006 ± 0.005	0.141 ± 0.197	0.006 ± 0.005
Cadmium				1.18 ± 0.72	0.24 ± 0.32	0.24 ± 0.32	1.98 ± 4.757	1.16 ± 1.36
Cobalt	0.0094	0.001-0.01	0.038					
Copper	0.12	0.13						
Cyanide								
Lead				0.064 ± 0.086	0.0313 ± 0.002	0.0313 ± 0.002	0.017 ± 0.0016	0.036 ± 0.051
Manganese	0.029	0.029-0.03	0.12					
Mercury				00.47 ± 0.059	0.02 ± 0.02	0.02 ± 0.02	0.13 ± 0.11	0.02 ± 0.01
Nickel	0.019	0.019-1.9	0.076	0.97 ± 1.23	1.56 ± 3.29	1.56 ± 3.29	0.83 ± 1.01	0.73 ± 0.71
Silver	0.15	0.15						
Vanadium								
Zinc	0.4	0.4						
Americium-241								
Cesium 137	0.01	0.0004-0.078	0.15					
Cobalt 60	0.0094	0.001-0.01	0.038					
U-234, U-235, U-236	0.002	0.01						

A comprehensive listing of Bv values for all elements in the periodic table was developed in Baes et al. (1984). This list has been used by risk assessors, often with little regard to the quality of the information, and the uncertainties associated with the values. The Bv values presented by Baes et al. (1984) were developed using a combination of published literature, correlation with other parameters, elemental systematics, or comparisons between observed and predicted concentrations in foods. According to the authors, the analysis of the literature values relied on a subjective evaluation of the experimental techniques and reliability of reported data, which could have caused an error in the final estimate. For example, where the reported soil and plant data were graphically presented in the original articles, the authors estimated the Bv values.

For elements with little or no experimental information, elemental systematics were used to derive best estimates of Bv. The authors extrapolated values from the trends determined from relationships established between concentrations factors for an element and for other adjacent elements on the periodic table. The implication in this exercise is that elements belonging to the same groups and families act similarly in the soil-plant environment.

Baes et al. (1984) also attempted to divide reported values into vegetative (leaves and stems) and nonvegetative (fruits, seeds, and tubers) plant parts, but did not clearly explain how the different Bv values were determined from data for whole plants only. Based on the values from the literature, Baes et. al. calculated geometric means for each element and reference, and constructed a distribution for Bv. The geometric means of these interference distributions were reported as the best estimates of Bv by Baes et al. (1984).

It appears that several of the assumptions used in developing the transfer factors are overly simplistic. The grouping of all edible plants into reproductive or vegetative parts with no regard to species differences in uptake seems counter-intuitive, since different species and plant parts tend to bioaccumulate minerals at different rates. Where data did not exist for one element, a value was extrapolated based on information for a chemically equivalent element. However, this too appears to be an oversimplification of the element-specific uptake behavior of plants.

Belcher and Travis (1989) presented chemical-specific distributions for BTFs along with corresponding means and standard deviations. These distributions were presented for different produce groups, of which only leafy vegetables, root vegetables, garden fruits, and fresh legumes are relevant to the EFPC food chain ingestion scenario. The Bv distributions presented by Belcher and Travis (1989) are based in large part on the data that was collected for the EPA (1985c) series on constituents of municipal sludge, and supplemented by data presented by Baes et al. (1984) and other secondary references. Belcher and Travis assumed a lognormal distribution and presented Bv values for each produce group in terms of means, standard

deviations, and ranges of untransformed data. The number of observations shown in association with each set of Bv values refers to the number of summary values extracted from the compendia (e.g., EPA 1985c) rather than the number of replicates and/or number of different plant types. A wide variety of soil conditions, chemical forms, and data quality were used in developing Bv values in the source studies.

At the recommendation of ORNL CRAC, the Bv values from NCRP (1989) were used in the EFPC BRA to estimate concentrations of inorganic chemicals and radionuclides in produce. ORNL has recommended use of these Bv values principally for screening-level risk assessments. The NCRP values have been used in the BRA and were supplemented with data (i.e., probability distributions) from the Belcher and Travis (1989) study. Where chemical-specific values for certain chemicals are not available from the NCRP documents, values developed by Baes et al. (1984) were used. Bv values for the reproductive plant parts were assumed to apply to roots and fresh legumes, while Bv values for vegetative plant parts applied to leafy vegetables.

At the recommendation of ORNL, the biotransfer coefficient for organic chemicals was calculated using the octanol-water partition coefficient (K_{ow}) and the equation presented in NCRP (1991):

$$Bv_{(dry\ wt.\ pasture)} = 10^{(1.588-(0.578 \log K_{ow})} \quad (15)$$

where

$$Bv_{(Fresh\ vegetables)} = 0.25\ Bv_{(pasture)} \quad (16)$$

The relationship between Bv and log K_{ow} was validated for a range of organic chemicals and originally presented by Travis and Arms (1988). The conversion of Bv for dry weight pasture to fresh vegetation (or produce) was presented as a default in NCRP (1991).

The Bvs that are provided in terms of plant dry weights will need to be converted to a wet-weight basis. This is necessary because the exposure equation is based on ingestion rates for wet weights of produce. This conversion is accomplished simply using the default water content of each produce group:

WRONG!

$$Bv_{WW} = Bv_{DW} \times CF\ \frac{g\ WW}{g\ DW} \quad (17)$$

Should be:

$$CF\ \frac{g\ DW}{g\ WW}$$

$$\frac{\frac{mg\ Hg}{kg\ plant\ (dry)}}{\frac{mg\ Hg}{kg\ soil\ (dry)}} \times \frac{kg\ plant\ (dry)}{kg\ plant\ (wet)} = \frac{\frac{mg\ Hg}{kg\ plant\ (wet)}}{\frac{mg\ Hg}{kg\ soil\ (dry)}}$$

Dry-weight to wet-weight conversions are available for various fruits and vegetables and for the exposed and protected produce categories in general (Baes et al. 1984). Belcher and Travis (1989) used the Baes et al. (1984) data and wet-weight ingestion rates presented in Yang and Nelson (1986) to develop dry-weight ingestion rates for six produce categories (i.e., leafy vegetables, garden fruit, fresh legumes, root vegetables, potatoes, and dry legumes). The verification of these values is complicated by the fact that Yang and Nelson (1986) only provided ingestion rates for three produce groups (i.e., leafy, exposed, and protected). Nevertheless, by aggregating produce categories from the Belcher and Travis (1989) study, it is possible to calculate dry-weight to wet-weight conversion factors for each produce group of interest (Table 5.9). The calculated conversion factors compare favorably with those presented by Baes et al. (1984) for exposed and protected produce.

Table 5.9. Wet weight to dry weight conversion factors based on Yang/Nelson vs. Belcher/Travis

Food Group	Belcher and Travis Components	Belcher and Travis Mean DW Intake	Yang and Nelson Mean WW Intake	Conversion Factor (WW/DW)	Baes Conversion Factors
Leafy	Leafy vegetable	2.61	39.3	15.02	na
Exposed	Fresh legume, garden fruit	11.8	86	7.29	0.13 7.94
Protected	Root, potatoes	36.6	150.4	4.11	0.22 4.50

Produce ingestion rates for use in exposure assessments have been compiled by a number of authors (e.g., Belcher and Travis 1989, Travis et al. 1983, Hoffman and Baes 1979, EPA 1989b, Yang and Nelson 1986). Default produce ingestion rates recommended by EPA (1990d) are 140 g wet weight of fruit per day and 200 g wet weight of vegetables per day. EPA recommends use of these values in conjunction with different percentage rates to estimate fractions of homegrown produce of total dietary fruit and vegetable intake. EPA notes that while it is more likely for farm families to cultivate fruits and vegetables, it is not necessarily true that they would be able to grow a sufficient variety to meet all of their dietary needs and tastes (EPA 1991a).

Total produce ingestion rates have been presented in terms of dry weight by Belcher and Travis (1989) and EPA (1990d). These dry-weight values must be converted to wet weights for calculating exposures. As discussed above, many problems are associated with the literature values for dry weight to wet weight conversion factors. The produce ingestion rates presented by

Belcher and Travis (1989) are 45.5 to 56.1 g dry weight per day, or approximately 246.4 to 305 g wet weight per day, as calculated using dry weight to wet weight conversion factors (Table 5.10). Ingestion rates of various fruit and vegetable groups for children and for the total U.S. population are presented in EPA (1991a) and shown in Table 5.11. However, the ingestion rates for produce are questionable because fresh legume ingestion rates appear to be set at an unrealistically high level that is 82% of the total dry weight produce ingestion.

Table 5.10. Produce ingestion rates for adults

Produce	Mean (g DW/day)	Range (g DW/day)	Range[a] (g WW/day)
Leafy Veg	2.6	2.4 - 3.0	36.5 - 45.06
Fresh Leg	0.8	0.6 - 1.1	4.37 - 8.02
Garden Fruit	11.0	9.7 - 12.0	70.71 - 87.48
Root Veg	3.3	3.0 - 3.6	12.33 - 14.80
Potatoes	30.0	27.4 - 32.0	112.61 - 131.52
Dry Legume	3.3	2.4 - 4.4	9.86 - 18.08
Total	51	45.5 - 56.1	246.38 - 304.96

[a] Wet weight range determined by multiplying dry weight range by appropriate conversion factor.

Source: Belcher and Travis 1989.

Table 5.11. Produce ingestion rates for children (age 7 to 12) and U.S. population (g DW/kg BW/day) and (g DW/day)

	Age 7 - 12 (30.5 kg)[a]		U.S. Pop (70 kg)[a]	
Produce	Mean (50th percentile)	90 - 95th percentile	Mean (50th percentile)	90 - 95th percentile
Leafy Veg	0.009 / 0.27	0.06 / 1.83	0.008 / 0.56	0.06 / 4.2
Fresh Leg	0.69 / 21.05	1.7 / 51.85	0.44 / 30.8	1.4 / 98
Root Veg	0.03 / 0.92	0.13 / 3.97	0.02 / 1.4	0.11 / 7.7
Garden Fruit	0.11 / 3.79	0.38 / 1.31	0.07 / 4.9	0.30 / 21
Total	0.839 / 26.03	2.15 / 58.96	0.54 / 37.16	1.87 / 130.9

[a] Body weight is mean of age group 6 to 9 (25 kg) and 9 to 12 (36 kg) for children and default body weight for adults.

Source: EPA 1989b.

EPA does not provide default produce ingestion rates for all age groups of concern. A study by Yang and Nelson (1986) provides the additional details needed for the BRA, including produce ingestion rates for different age groups. These values are based on the same survey described by EPA (1989b). Yang and Nelson (1986) have organized the data into major food classes rather than the specific food products considered in the original survey. This reorganization results in slight differences from the values presented in EPA (1989b). The Yang and Nelson (1986) study has the advantage of presenting a much more detailed data set that is not limited to adult receptors as is the information presented in EPA (1989b, 1991a). Wet weight produce ingestion rates for adults range from 270.6 to 303.1 g per day (Table 5.12), which is comparable to the total produce ingestion rates recommended by EPA (1990c).

Table 5.12. Yang and Nelson mean adult produce ingestion rate data (g WW/day ± standard error)

Food Class	U.S. Population	North East	North Central	South	West
Leafy	39.2 ± 3.5	38.1 ± 1.5	37.1 ± 1.5	38.4 ± 1.2	45.3 ± 1.8
Exposed	86.0 ± 1.5	88.5 ± 3.0	87.8 ± 2.9	76.9 ± 2.4	95.5 ± 3.6
Protected	150.4 ± 2.3	137.2 ± 4.5	150.1 ± 4.3	160.1 ± 3.6	152.5 ± 5.3
Total Produce	282.6 ± 3.5	270.6 ± 6.9	282.4 ± 6.7	280.7 ± 5.6	303.1 ± 8.2

Source: Yang and Nelson 1986.

The EPA EFH (EPA 1989b) lists daily consumption rates of particular fruits and vegetables for different percentile groups in the U.S. population. However, no information is provided on total ingestion of the different fruits and vegetables. The ratios of the 50th to 95th percentile ingestion rates for the three groups of produce listed in the EFH (i.e., raw vegetables, cooked vegetables, and fruits) are 0.27, 0.32, and 0.32, respectively. The mean of the three groups is 0.3, which suggests that the 50th percentile group ingests one-third as much produce as the 95th percentile group. Based on this limited data set, the MLE ingestion rate for adults is calculated as one-third (the ratio of the 50th to 95th percentiles) the RME value.

The RME ingestion rate that is used in this assessment is equal to the total produce ingestion rate of 340 g/d [i.e., 200 g vegetables/d and 140 g fruit/d presented in EPA (1989b, 1991c)]. The adult MLE value is calculated to be one-third of the RME, or 113 g/d. The produce ingestion rates for children are calculated using ingestion rates for the nearest match age group presented by Yang and Nelson (1986). The RME value for children is calculated by multiplying the age-specific ingestion rate by the ratio of the adult RME to adult ingestion rate presented by

Yang and Nelson. The MLE value for children is similarly calculated using the adult MLE value.

As developed, the produce ingestion pathway assumes that residential and agricultural receptors grow fruits and vegetables in a home garden. It is assumed that only a small fraction of total fruit and vegetable intake is from the home garden. Exposure assessment for produce ingestion requires an estimate of the proportion or fraction of ingested produce that originates in the contaminated area. The proportion is assumed to consist of the fraction of homegrown fruit and homegrown vegetables for residential exposure and exposure in an agricultural setting (EPA 1989a,b; 1991a). Values of 35 and 23% have been adopted for RME and MLE estimates, respectively. The RME value is the average of the recommended fruit fraction ingested and the vegetable fraction ingested presented in EPA (1989e).

For residential receptors, the value for exposure frequency used to calculate the RME and MLE is based on the assumption of daily ingestion of produce that is available only during the normal growing season (i.e., June through October). For agricultural receptors, the RME value is based on the assumption that produce is preserved during the growing season, and is available daily, throughout the year. The MLE value for exposure frequency among agricultural receptors is the same as residential receptors. The values for the exposure duration, body weights, and averaging times are the same as presented for previous exposure pathways.

EFPC Vegetation and Garden Produce Study. The EFPC vegetation and garden produce study was conducted to examine the transfer of contaminants in EFPC floodplain soils to garden crops and floodplain vegetation. Mercury is the principal contaminant of concern in the EFPC system. This study focused on the evaluation of toxic metals in plant tissue. A complete overview of the sampling and analysis plan for this study is contained in the supplemental document *Phase Ib Sampling and Analysis Plan Addendum for Garden Produce and Vegetation* (Radian 1993a). This section of the BRA provides an overview of the purpose and objectives of the study, and an assessment of the results.

Metals may be transferred from soils to plant tissue via root uptake. Historical studies of EFPC biota indicate the presence of chemical contaminants originating from floodplain soils (Gist 1987; Van Winkle et al. 1984). The rate of uptake and accumulation of chemicals in plant tissue is specific for each chemical and plant species under evaluation and also depends on the physical and chemical properties of the surrounding soil environment. In the absence of monitoring data, food chain modeling (as described above) is used to "move" the contaminants from the soil matrix to vegetation, beef, and dairy products and then to humans. These models make use of conservative BTFs that have been derived from a limited data set. Use of the

published BTFs often results in extremely conservative upper-bound estimates that do not reflect the true potential for accumulation. Therefore, site-specific information is needed to provide better informed and more reasonable values for Bvs. The results of the vegetation sampling and analysis program have been used to refine estimates of food chain transfer and to support a quantitative analysis of uncertainty.

The study involved sampling and analysis of floodplain grasses and garden vegetation that were planted in experimental plots. Garden crops were chosen to represent types of foods typically raised and consumed by home gardeners. Floodplain grasses and browse may be consumed by cattle (and other terrestrial mammals), which in turn may become a source of contaminant exposure (i.e., via food) in humans. Floodplain grasses along with composite soil samples were taken at three locations along surveyed north-south transects and at the Phase Ia reference site on Hinds Creek. The three locations on EFPC are in the vicinity of Bruner Center, on the Green property farm, and on the Jackson property farm.

Garden vegetable crops were grown in two garden plots: the Bruner site and a reference site in the home garden of Dr. C.T. Hadden, a Science Applications International Corporation (SAIC) scientist and resident of Oak Ridge. Crops of kale, beets, and tomatoes were planted. A total of 18 composite soil samples were obtained prior to planting the seeds or plants. Samples of fruits, leaves, roots, and stems were obtained from the garden plots.

All samples were analyzed by NAA. Analyses for vegetation were conducted by the Nuclear Services Center at North Carolina State University and soil samples were analyzed by ORNL. Concentrations of different metals were reported in terms of mg/kg dry weight in soil, and mg/kg wet weight in plant tissue.

The concentrations of inorganic contaminants in vegetation and associated soil samples are provided in Table 5.13. A regression analysis was conducted using the least squares procedure to examine the relationship between contaminant concentration in soil and in plant tissues. Two sets of regressions were generated for each contaminant-plant pair. In one analysis, the y intercept was calculated, and in the second analysis, the regression line was forced through the origin. Figure M.1 in Appendix M shows the results of the regression analyses. The data used in the regression analyses are presented in Table M.2. As determined by the r^2 values, the regressions generally showed poor correlation between soil and plant concentrations. Notable exceptions are cadmium concentrations in tomato, and chromium concentrations in beet and tomato.

There are several possible reasons for the low correlations observed. Foremost is the issue of heterogeneity in contaminant levels in soil samples. Ideally, the experimental garden study

Table 5.13. Chemical concentratios in soil and crop tissue (mg/kg wet weight)

Plant and Sample ID	Chemical	Measured Concentrations	
		Soil	Vegetation
	Arsenic		
BEET			
VG5312470		7.9	0.070
KALE			
VG0002529		9.5	0.034
VG5312519		8.2	0.062
VG5312569		7.3	0.010
TOMATO			
VG5312747		8.9	0.008
	Cadmium		
BEET			
VG5312468		8.4	0.686
KALE			
VG5312521		6.2	0.517
VG5312533		6.7	0.521
VG5312538		6.7	1.564
VG5312545		8.5	0.900
VG5312557		6.9	0.407
TOMATO			
VG5312711		5.8	0.160
VG5312723		6.3	0.108
VG5312735		5.5	0.137
VG5312747		8.4	0.257
	Chromium		
BEET			
VG5312456		51.6	0.473
VG5312468		56.2	0.173
VG5312468		56.2	0.145
VG5312470		48.4	0.514
KALE			
VG0002517		16.3	0.061
VG0002529		32.4	0.164
VG0002531		24.7	0.514
VG5312519		61.6	0.552
VG5312521		47.9	0.122
VG5312545		49.6	0.074
VG5312569		46.9	0.196
TOMATO			
VG5312711		54.5	0.107
VG5312711		54.5	0.105
VG5312723		43.8	0.047
VG5312747		41.5	0.051

5.15

Table 5.13. (continued)

Plant and Sample ID	Chemical	Measured Concentrations (dry) Soil	Measured Concentrations (wet) Vegetation
	Mercury		
BEET			
VG5312456	4.11	273	1.082
VG5312468 root		171	0.631
VG5312468		171	0.765
VG5312470		196	2.720
KALE			
VG5312519		204	3.198
VG5312521		188	0.348
VG5312533 leafy	15.02	141	0.170
VG5312533		141	0.309
VG5312545		270	0.180
VG5312557		237	0.133
VG5312569		699	1.278
TOMATO			
VG5312747 fruit	7.29	236	0.416
	Zinc		
BEET			
VG5312456		131	6.505
VG5312468		144	8.361
VG5312468		144	7.460
VG5312470		138	11.232
KALE			
VG0002517		35.9	8.223
VG0002529		37.6	4.203
VG0002531		36	5.497
VG5312519		173	6.488
VG5312521		195	8.037
VG5312533		207	12.390
VG5312533		207	11.189
VG5312545		147	9.270
VG5312557		132	7.339
VG5312569		165	8.210
TOMATO			
VG5312711		174	3.037
VG5312711		174	1.684
VG5312723		193	2.078
VG5312735		167	2.749
VG5312747		170	5.837
VG0002719		384	1.092
VG0002721		272	1.092

Calculated plant concentrations were determined using BTFs converted to a wet weight basis.
Dry weight to wet weight conversion factors are presented in Table 5.9.

should be conducted using a known series of uniformly contaminated soil samples. This would be difficult to accomplish in a laboratory setting, and was impossible in the field study. Soil samples were obtained from the floodplain prior to planting the seeds for the experimental study. Although the study was conducted at a site known to be contaminated with high levels of mercury, considerable variability remains in observed concentrations, even in samples obtained within 1 foot of each other. Other reasons for the lack of correlation include the limited number of data points, the need for a wider range of soil concentrations, and the attempt to fit a straight line on data that may not have been linear. In the majority of cases, only four to seven observations were obtained from the field study, and in most cases, these data points were based on one or two soil concentrations. If the data were nonlinear, regression lines drawn using log-transformed data would be expected to result in greater r^2 values than with the untransformed data. This was not the case with the garden study data, and suggests that more data points (with a wider range of soil concentrations) were required for this analysis.

The slope of the regression is equivalent to the B_v. The slopes were compared to B_v values obtained from literature for similar plant groups. Kale was compared to B_vs for leafy vegetables, beets were compared to root vegetables, and tomatoes were compared to garden fruits. In addition, calculated chemical concentrations using B_vs from literature were compared to the measured concentrations in plants. The validity of this comparison is questionable, since the garden study data were based on a limited number of plants, while literature values are based on the results of several studies and reflect bio-uptake by morphologically similar plant parts grouped across several species. Nevertheless, experimentally determined B_vs were generally much lower than the literature values (Table 5.14). Furthermore, chemical concentrations in plants from the EFPC garden study were generally one to two orders of magnitude lower than the equivalent concentrations calculated using the B_v values from NCRP (1991) (Table M.2). The calculated concentrations that most closely matched observed chemical concentrations in the produce were obtained using the B_v values from Baes et al. (1984). This further suggests that the B_v values obtained from the NCRP publications (recommended by ORNL) are very conservative.

Ingestion of Home-produced Meat

This pathway evaluates exposure of farm families to beef obtained from cattle raised on land in the EFPC floodplain. The "home-produced" meat ingestion pathway is assumed to be relevant only for the homesteader scenario. Data on chemical concentrations in livestock raised on, or grazing in, the vicinity of the EFPC floodplain are limited. The primary mechanisms of exposure of cattle is assumed to be inadvertent direct ingestion of soil and ingestion of forage crops growing in floodplain soils and ingestion of surface water from the creek. Grain eaten by beef cattle is assumed to be free from contamination.

Table 5.14. Comparison of Bv values from field study and from the literature

Chemical	Plant	Observed Slope	Literature Values for Bvs		
			NCRP-20	Belcher and Travis 1989	Baes et al. 1984
Arsenic	Kale	0.009	0.08	0.041	0.003
Cadmium	Kale	0.088	0.5	0.079	0.037
	Tomato	0.042	0.5	0.146	0.033
Chromium	Beet	-0.049		0.017	0.001
	Kale	0.003		0.003	
	Tomato	0.005		0.004	
Mercury	Beet	0.002	0.3	0.029	0.044
	Kale	0.001	0.3	0.003	0.06
Zinc	Beet	0.065	0.4		0.2
	Tomato	-0.012	0.4		0.2
	Kale	0.024	0.4		0.1

Note: Literature values were converted to wet-weight basis by dividing the dry-weight based values by 15.02 for leafy plants, 4.5 for roots, and 7.94 for exposed produce or fruits (Table 5.9).

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The following equation will be used to estimate chemical intake from the ingestion of meat:

$$Intake = C_{beef} \times \frac{IR \times RAF \times FI \times EF \times ED}{BW \times AT} \quad (18)$$

where

C_{beef} = chemical concentration in animal tissue (beef),
IR = ingestion rate of meat by humans (kg WW/d),
RAF = relative absorption factor (unitless),
FI = fraction ingested from contaminated area (unitless),
EF = exposure frequency (d/year),

ED = exposure duration (years),
BW = body weight (kg),
AT = averaging time (days).

In the absence of monitoring data, equilibrium concentrations of contaminants in cattle are calculated using a variation of the food chain model described in EPA (1990d) and Belcher and Travis (1989). The concentration of contaminants in animal tissue is assumed to be a function of the concentration of chemicals in soil, forage crops, and surface water, and is calculated as follows:

$$C_{beef} = Ba \times [(Cp \times Qp) + (Cs \times Qs) + (Cw \times Qw)] \quad (19)$$

where

Ba = biotransfer factor from feed to beef tissue (d/kg),
Cp = chemical concentration in the forage (mg/kg DW),
Qp = forage ingestion by the beef cattle (kg DW/d),
Cs = chemical concentration in soil (mg/kg),
Qs = soil ingestion rate by the beef cattle (kg/d),
Cw = chemical concentration in water (mg/L),
Qw = water ingestion by the beef cattle (L/d).

Data are available on chemical concentrations in floodplain grasses and browse. This information has been used to supplement the results of food chain modeling. The results for floodplain grasses may be used as a surrogate for forage crops. These values have been used along with estimates of levels in forage based on the assumption of equilibrium partitioning between soils and vegetation. Monitoring data are available to determine chemical concentrations in soil and surface water.

Values for soil, forage, and water ingestion rates by beef and milk cows have been developed in a number of sources. Travis and Arms (1988) used default dry feed ingestion rates of 16 kg dry weight per day for lactating cows, and 8 kg dry weight per day for beef and nonlactating cattle. Belcher and Travis (1989) determined the quantity of forage eaten by beef cattle as 3.6 kg dry weight per day, and by dairy cows as 10.37 kg dry weight per day. The quantity of soil ingested by beef and dairy cows was identified as 0.39 and 0.41 kg/d, respectively. Because these values differentiate between dairy and beef cattle, they are used in the EFPC food chain exposure model.

Water consumption rates by beef and dairy cows were not addressed in the food chain models described above (Travis and Arms 1988, Belcher and Travis 1989). Water ingestion rates obtained in the literature averaged 104 L/d for dairy cattle and 35 L/d for beef cattle (Osweiler et al. 1985; Healy 1986; Kutches 1977). These values have been used in the EFPC food chain exposure model.

The B*a* is defined as the equilibrium concentration of pollutant in an organism or tissue (mg/kg) divided by the average daily intake of pollutant (mg/d) (Belcher and Travis 1989). The B*a* differs from the BCF, which is the traditional measure of a chemical's potential to accumulate. The BCF is defined as the equilibrium concentration of pollutant in an organism's tissue (μg/g) divided by the equilibrium concentration of pollutant in food (μg/g), while the B*a* is equal to the BCF divided by the animal's daily food intake.

Belcher and Travis (1989) provided BTFs for inorganics to milk (B*m*) and beef muscle tissue (B*a*). These factors were based on published BCFs divided by a default daily dry feed ingestion rate for the given animal group. The dry feed ingestion rates used were 16 kg/d for lactating cows and 8 kg/d for nonlactating cows or beef cattle (Travis and Arms 1988).

Other sources of data for B*a*s include NCRP (1989, 1991) and Baes et al. (1984). These B*a*s are largely based on data collected by Ng et al. (1977) and Ng (1982). Table 5.15 shows the range of values for B*a*s obtained from these sources. At the recommendation of ORNL, B*a* values for beef muscle presented in NCRP (1989) are used. Where chemical-specific values are not available from those sources, the values presented in Baes et al. (1984) are used.

As recommended by ORNL, the B*a*s for organic chemicals (i.e., transfer to beef muscle tissue) are calculated based on the relationship of B*a*s to the octanol-water partition coefficient (K_{ow}) presented in NCRP (1991) based on the equation derived by Travis and Arms (1988):

$$Ba = 10^{(-7.6 + \log Kow)} \qquad (20)$$

Meat ingestion rate data are available from various literature sources. However, each source does not necessarily contain the full information needed for this assessment (e.g., data are available for adults but not children; data are available for the MLE estimate but not the RME estimate). The available data are summarized in Table 5.16.

The EPA supplemental guidance (EPA 1991a) value of 100 g/d was chosen as the adult RME beef ingestion rate because it is derived from the most recent EPA guidance. This value

Table 5.15. Biotransfer factors from soil to beef muscle tissue

Chemical	NCRP 1989		NCRP 1991		Baes et al.		Ng et al.	
	Milk (d/L)	Beef (d/kg)	Milk (d/L)	Beef (d/kg)	Milk (d/kg)	Beef (d/kg)	Beef (d/kg) Median	Beef (from data summary)
Aluminum			0.0002	0.0005	0.0002	0.0015		
Arsenic			0.0001	0.02	0.00006	0.002		
Barium	0.0004	0.0002	0.0005	0.0002	0.00035	0.00015	0.000097	0.000097
Beryllium			0.000002	0.005	0.0000009	0.001		
Cadmium			0.002	0.001	0.001	0.00055		0.00035
Cobalt	0.002	0.03	0.002	0.03	0.002	0.020	0.0097	0.002 to 0.069
Copper			0.002	0.01	0.0015	0.010	0.013	0.009
Cyanide								
Lead	0.0003	0.0008	0.0003	0.0008	0.00025	0.0003		0.0004
Manganese	0.0003	0.001	0.0003	0.001	0.00035	0.0004	0.00039	0.0005
Mercury	0.0005	0.01	0.0005	0.01	0.00045	0.25		
Nickel	0.001	0.002	0.02	0.005	0.001	0.006	0.002	0.002
Silver	0.01	0.005	0.006	0.003	0.020	0.003	0.0019	0.002
Vanadium			0.0005	0.01	0.00002	0.0025		
Zinc	0.01	0.1	0.01	0.1	0.01	0.10	0.12	0.098
Americium-241	0.0000004	0.00002	0.000002	0.00005	0.0000004	0.0000035		
Cesium 137	0.008	0.03	0.01	0.05	0.007	0.020		
Cobalt 60	0.002	0.03	0.002	0.03	0.002	0.020	0.0097	
U-234, U-235, U-236	0.0006	0.01	0.0004	0.0008	0.0006	0.0002		

Table 5.15. (continued)

Chemical	Ng et al.		Belcher and Travis	
	Beef (from NRC) (d/kg)	Beef (from other sources) (d/kg)	Beef (d/kg) (based on dry feed ingestion rate)	Milk (d/kg) (based on dry feed ingestion rate)
Aluminum				
Arsenic			0.0072 ± 0.0035	0.0003 ± 0.0002
Barium	0.0032	9.7E-5 - 5.0E-4		
Beryllium			0.002 ± 0.002	3E-6 ± 3E-6
Cadmium			0.00 ± 0.005	0.0055 ± 0.002
Cobalt	0.013	0.001 - 0.017		
Copper	0.008	2E-3 - 0.013		
Cyanide				
Lead			0.006 ± 0.006	0.006 ± 0.006
Manganese	0.0008	3.8E-4 - 0.005		
Mercury			0.44 ± 0.52	0.02 ± 0.02
Nickel	0.053	0.001 - 0.0067	0.021 ± 0.015	0.006 ± 0.002
Silver	0.017	0.001 - 0.022		
Vanadium				
Zinc	0.03	0.002 TO 0.12		
Americium-241				
Cesium 137	0.004	0.014-0.03		
Cobalt 60	0.013	0.001-0.017		
U-234, U-235, U-236				

Table 5.16. Comparison of meat ingestion rates from different literature sources

Source	Age Group	MLE or RME	Ingestion Rate	Food Type	Comments
Human Health Evaluation Manual; Supplemental Guidance (EPA 1991)	Adults	RME	100 g/d	beef	The rate is actually 75 g per day; however, this assumes a 75% FI; without taking into account the FI, the rate is 100 g/d
RAGS (USEPA 1989a)		MLE	112 g/meal	beef	No guidance was given on the number of beef meals consumed per day
		RME	280 g/meal		
Yang and Nelson (1986)	Adults	MLE	87.6 g/d	beef	
			172.2 g/d	all meat	
	Children	MLE	127 g/d	all meat	No values were available for beef ingestion only
Breidenstein, undated	Adults	MLE	84 g/d	beef	

was used as the starting point for deriving the remaining ingestion rates. The rates were calculated as follows:

- The first step involved calculating an MLE ingestion rate for adult beef consumption. This was accomplished by taking the ratio of the 50th (MLE) to the 95th (RME) percentile beef ingestion rates from RAGS (112/280 = 0.4) and multiplying it by the adult RME beef ingestion rate of 100 g/d. This yields an adult MLE beef ingestion rate of 40 g/d.
- Yang and Nelson's total meat ingestion rates for children were then converted to beef ingestion rates. As shown in Table 5.16, Yang and Nelson provide adult ingestion rates for both beef and total meat, but ingestion rates for children include only values for total meat. The conversion to beef ingestion rates for children was accomplished by taking the adult ratio of beef to total meat (87.6/172.2 = 0.51) and multiplying by the children's total meat ingestion rates (0.51 × 127 g/d = 64.6 g/d).
- The last step was to derive MLE and RME beef ingestion rates for children using the ratios obtained from the Yang and Nelson values (calculated above) and then applying them to the EPA adult ingestion rates. The ratio of the children to the adult beef ingestion rates (e.g., for children 3 to 12 years: 64.6/87.6 = 0.74) multiplied by the

adult RME yields the RME value for children (0.74 × 100 g/d = 74 g/d). Likewise, multiplying this ratio by the adult MLE provided the children's MLE value.

The fraction of meat ingested that originates from animals raised in the contaminated area is assumed to be 75% for the RME. This value is in agreement with guidance presented in EPA (1991a). For the MLE, the fraction ingested is assumed to equal the proportion of home-raised beef ingested by the average farm household, as presented in EPA (1989b). Since ingestion rate values were based on average daily intake, exposure frequency is 350 days per year. Exposure duration, body weights, and averaging times are the same as those used in the evaluation of other exposure pathways.

Ingestion of Home-produced Milk/Dairy Products

Home-produced dairy products include milk and milk products, such as cheese, butter, and cream. This exposure pathway focuses only on consumption of milk potentially contaminated with chemicals originating from EFPC soils. Ingestion of potentially contaminated home-produced dairy products is assumed to be relevant only for families living in an agricultural setting. This exposure pathway is based on the assumption that the milk-producing cows owned by the farm families graze on the contaminated EFPC floodplain. Currently, no farm families in EFPC are consuming milk and dairy products obtained from cattle raised on EFPC floodplain soils. Therefore, this pathway is considered only as a future exposure scenario.

Chemical intake is calculated using the following formula:

$$Intake = C_{milk} \times \frac{IR \times RAF \times FI \times EF \times ED}{BW \times AT} \qquad (21)$$

where

C_{milk} = chemical concentration in animal tissue (milk),
IR = ingestion rate of milk by humans (kg WW/d),
RAF = relative absorption factor (unitless),
FI = fraction ingested from contaminated area (unitless),
EF = exposure frequency (d/year),
ED = exposure duration (years),
BW = body weight (kg),
AT = averaging time (days).

In the absence of monitoring data, equilibrium concentrations of contaminants in milk are calculated using methods described in Belcher and Travis (1989) and Travis and Arms (1988). The methods are the same as those presented above for ingestion of beef. However, concentrations in milk are based on the use of biotransfer factors for dairy rather than beef. Contaminant biotransfer values for feed to milk have been derived from a number of sources (Table 5.15).

The equation for calculating contaminant concentration in milk is a function of the concentrations of each chemical in soil, forage crops, and surface water, and is calculated as follows:

$$C_{milk} = Bm \times [(Cp \times Qp) + (Cs \times Qs) + (Cw \times Qw)] \quad (22)$$

where

Bm = biotransfer factor from feed to milk (d/kg),
Cp = chemical concentration in the forage (mg/kg DW),
Qp = forage ingestion by milk cattle (kg DW/d),
Cs = chemical concentration in soil (mg/kg),
Qs = soil ingestion rate by milk cattle (kg/d),
Cw = chemical concentration in water (mg/L),
Qw = water ingestion by milk cattle (L/d).

Data on quantities of feed, soil, and water eaten by cattle also have been discussed above. Milk cows will be assumed to ingest 16 kg dry weight per day (Travis and Arms 1988) to 10.37 kg dry weight per day of dry feed, and 0.41 kg/d of soil (Belcher and Travis 1989). An ingestion rate of 104 liters of water per day for dairy cattle will be used. This value is an average of values presented in Osweiler et al. (1985), Healy (1986), and Kutches (1977).

Belcher and Travis (1989) provided Bms for inorganic chemicals in milk. However, the values of those Bms were based on published BCFs divided by an assumed dry feed ingestion rate for the given animal group. The dry feed ingestion rates used in the calculation were 16 kg/d for lactating cows (Travis and Arms 1988). Other sources of data for Bms include NCRP (1989, 1991) and Baes et al. (1984), which are largely based on data collected by Ng et al. (1977) and Ng (1982). Table 5.15 shows the range of possible values for Bms for milk obtained from these sources.

At the recommendation of ORNL, the Bm values for inorganic chemicals in milk presented in NCRP (1989) have been used in the assessment of EFPC. Where chemical-specific values are not available from that source, the values presented in Baes et al. (1984) have been used. Bms

for organic chemicals in milk have been calculated based on the relationship of Bms to the octanol-water partition coefficient (K_{ow}) presented in NCRP (1991) and developed by Travis and Arms (1988):

$$Bm = 10^{(-8.10 + \log Kow)} \quad (23)$$

Milk and dairy ingestion rates are presented in the EFH (EPA 1989e, 1990b). The EFH recommends a 90th percentile milk ingestion rate equivalent to 400 g/d and an average consumption rate equivalent to 160 g/d. These values are used for the adult RME and MLE milk ingestion rates.

Milk and dairy ingestion rates for children are not provided by EPA. Milk ingestion rates are presented by Yang and Nelson (1986) for age groups <1 year to 60+ years. These rates are used to determine RME and MLE milk ingestion for children potentially exposed at EFPC. The milk ingestion rate for each children's age group is first multiplied by the RME or MLE adult ingestion rate presented in EPA (1990b). This value is then divided by the adult milk ingestion rate presented by Yang and Nelson to determine the RME or MLE value for the specific children's age group.

Ingestion rates for milk and dairy products were presented in EPA (1989b) and Yang and Nelson (1986) in units of mass per day rather than the expected volumetric units of liters per day. The density of milk ranges from 1.028 to 1.035 kg/L (Baes et al. 1984); therefore, little error is introduced in defining the parameter in terms of kilograms rather than liters. Biotransfer factors for cattle feed to milk are presented in NCRP (1991) as days per liter, and by Baes et al. (1984) in terms of days per kilogram.

The fraction of dairy products ingested from home production is based on the recommendations presented in EPA (1989b, 1991a). The recent supplemental EPA guidance recommends a reasonable worst-case farm family home-produced dairy ingestion rate of 75%. This value is used for the RME fraction ingested value. EPA (1989b) reports an average farm family home-produced dairy consumption rate of 40%, which is used for the MLE fraction ingested value.

Since the ingestion rate values are based on average daily intake, exposure to home-produced milk and dairy products is assumed to occur daily, 350 days per year. Values for exposure duration, body weight, and averaging times are the same as for previously outlined pathways.

5.2.2.11 Ingestion of recreationally caught fish

Fishing in EFPC has been discouraged since the time contamination was observed in floodplain soils and sediments. Although notices have been posted on the creek, residents of Oak Ridge have access to the creek and may fish these waters. This exposure pathway has been included in the BRA based on the assumption that residents of Oak Ridge who make recreational use of EFPC will catch and ingest fish obtained from the creek. Fish may bioconcentrate or bioaccumulate COCs directly from the water or from ingesting contaminated prey or food items. Chemical concentrations in fish will be determined from historical records [Biological Monitoring and Abatement Program (BMAP) programs] and the results of the Phase Ib sampling program. Ingestion exposure has been calculated as follows:

$$Intake = C_{fish} \times \frac{IR \times RAF \times FI \times EF \times ED}{BW \times AT} \qquad (24)$$

where

C_{fish} = chemical concentration in fish tissue,
IR = ingestion rate of fish (kg/d),
RAF = relative absorption factor (unitless),
FI = fraction ingested from contaminated area (unitless),
EF = exposure frequency (d/year),
ED = exposure duration (years),
BW = body weight (kg),
AT = averaging time (d).

Recreationally caught fish are not consumed with the same frequency and quantity as market-purchased fish. The adult fish ingestion rates used in the evaluation of this pathway are based on values presented in EPA (1989b, 1991a). The recent supplemental EPA guidance presents an average consumption rate of 54 g/d, which is the 90th percentile value for recreational fishermen developed by Pierce et al. (1981, as cited in EPA 1989b). This value is used here for the adult RME ingestion rate. The 50th percentile recreational fishermen ingestion rate developed by Pierce et al. (1981) is 23 g/d. This value is used as the adult MLE ingestion rate. Since ingestion rates were determined as average daily ingestion, the exposure frequency is 350 days per year.

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Yang and Nelson (1986) present mean ingestion rates of fish and shellfish for age groups <1 to 60+ years. These values are used in the evaluation of the MLE and RME ingestion rates for children. The children's ingestion rates are calculated by multiplying the ingestion rate

presented by Yang and Nelson (1986) for the nearest match age group by the RME or MLE adult ingestion rate. This value is then divided by the adult ingestion rate presented by Yang and Nelson (1986).

Because of the low productivity of the creek, the proportion of recreationally caught fish originating in the creek is expected to be relatively low. The fraction of such fish is assumed to be 5% for the RME and 1% for the MLE. These values are based on professional judgment regarding activity of recreational fisherman in the Oak Ridge area.

5.2.3 Uncertainty in Exposure Assessment

Risk assessment should be viewed as an iterative process that proceeds through several levels of analysis and refinement. The exposure assessment component contributes largely to the need for this phased approach. It is clear from Sects. 5.2.1 and 5.2.2 that a broad range of information and analyses are required in conducting an exposure assessment. Although exposure data may be based on direct measurement (e.g., monitoring data), much of the information required to estimate intake or dose is based on assumptions, inferences, or professional judgment. Consequently, uncertainty analysis in exposure assessment is critical. It is needed to make explicit the limitations of the analysis and to facilitate an understanding of the reliability of the results.

This section of the BRA addresses uncertainty in the exposure assessment. An overall analysis of uncertainty in the risk assessment is presented in Sect. 5.5. Several types of uncertainty may be identified in exposure assessment (EPA 1992d):

- **Scenario Uncertainty:** missing or incomplete information needed to define the exposure scenario,
- **Model Uncertainty:** inadequate scientific theory or basis for exposure estimates or calculations, and
- **Parameter Uncertainty:** inadequate information to quantify an exposure variable or parameter.

All of these elements contribute to uncertainty in exposure estimates for EFPC human receptors. Often, the effect of scenario uncertainty and inadequate scientific models may be considered only qualitatively in a human health risk assessment. These contributors to uncertainty in the EFPC exposure assessment are discussed briefly below. Most of this section, however, addresses parameter uncertainty. Given that the exposure scenarios are meaningfully defined and the current limitations of scientific exposure models cannot presently be transcended (i.e., during the timeframe of this study), parameter uncertainty becomes the key focus of uncertainty analysis in risk assessment.

In the EFPC BRA, an attempt has been made to quantitatively examine the uncertainty surrounding the exposure variables (factors) used in the intake and dose equations. Rather than treating these factors as single point estimates, PDFs have been developed for use in Monte Carlo simulation. The methods of Monte Carlo analysis are described more fully in Sect. 5.5. Briefly, Monte Carlo analysis is a method that uses computer simulation to propagate the uncertainty inherent in the exposure assumptions through to the final estimates of risk to human health. Each uncertain variable must be characterized as a PDF. The final risk estimate is also a PDF and may be statistically evaluated with regard to the likelihood of exceeding a certain risk estimate.

In the following sections, three principal sources of uncertainty in exposure assessment are discussed. The last of these sections (parameter uncertainty) provides the rationale for the identification and selection of PDFs used in Monte Carlo simulation.

5.2.3.1 Scenario uncertainty

The basis for the exposure assessment is the identification and delineation of exposure scenarios. These scenarios are developed given an understanding of the nature and extent of contamination, the behavior of the COCs in the environmental media under investigation, and a knowledge of the receptors at potential risk of exposure. The EFPC risk assessment team endeavored early in the risk assessment process to establish consensus concerning potential exposure scenarios of importance in the BRA. The EFPC risk assessment team presented proposed exposure pathways to DOE, EPA Region IV, the State of Tennessee, and ORNL CRAC. Through a process of review and refinement, the exposure scenarios and pathways that are presented in this analysis were adopted.

The EFPC risk assessment staff is confident that the scenarios and pathways form a comprehensive basis for estimates of intake or dose. Uncertainty enters into the evaluation when pathways were not included in, or were eliminated from, the assessment, and when pathways have been incorrectly defined. The exposure pathways that were eliminated from the analysis include: inhalation exposure to entrained particulate matter in the residential setting; ingestion exposure to game (e.g., deer, quail, etc.); dermal exposure to contaminants during showering; direct (inadvertent) ingestion exposure of children to sediments; and use of EFPC as a source of irrigation water. All of these pathways were excluded because they were considered to be inconsequential to the overall estimate of intake or dose. Inhalation of organics volatilized from soils also was excluded from consideration because no VOCs were detected in the soil.

It is important also to consider uncertainty associated with pathways or scenarios that may have been incorrectly defined. In general, the risk assessment of EFPC attempted to follow the

conventions presented by EPA Headquarters in RAGS (EPA 1989a, 1991c). Where guidance was lacking, direction was obtained from ORNL CRAC, EPA Region IV, and from the published literature of risk assessment studies and methods. The open land use scenario is an example where guidance is limited. This scenario was defined based on an understanding of activities and potential behavior of residents of Oak Ridge living in the vicinity of EFPC. The open land use scenario was defined to include both recreation use of the open land surrounding EFPC as well as recreational use of the creek. In the final analysis, the design of these exposure assessments (i.e., for open land use) was based on professional judgment.

The combined influence of scenario uncertainty on the results of the EFPC human health risk assessment is considered very minimal. The effect of pathway exclusion (i.e., those noted above) is projected to influence the final risk estimates (i.e., combined risks across pathways) less than one order of magnitude.

5.2.3.2 Model uncertainty

Uncertainty in the final risk estimates is directly related to the models used in risk assessment to evaluate contaminant transport and transformation in the environment, estimate intake or dose, derive toxicity measures for risk assessment, and quantify risk based on the results of the exposure and toxicity assessments. This section focuses on the uncertainty in the exposure assessment. The models used to derive toxicity measures have not been considered other than to reacknowledge the existence of great levels of uncertainty associated with the toxicity measures used in risk assessment.

Uncertainty is associated with the models used to characterize environmental fate of contaminants. In the evaluation of EFPC, no surface water or groundwater transport models have been used in the human health risk assessment. Models have been used, however, to project concentrations in produce, beef, and dairy given a knowledge of contaminant concentrations in soil and an understanding of the physical/chemical transport parameters. Very limited guidance is available from EPA Headquarters or EPA Region IV in addressing food chain pathways. Working with ORNL CRAC, the EFPC risk assessment team established appropriate equations for modeling contaminant movement in the food chain. These equations are presented and discussed in preceding sections.

A considerable amount of uncertainty is associated with exposure estimates for the food chain pathways. The equations (or models) used for the produce pathway are fairly straightforward and a number of similar examples can be found in the literature. The equations for the beef and dairy pathways are more problematic, since less literature exists to set a precedent for

use of these algorithms in human health risk assessment of nonradiological contaminants. The EFPC BRA has made use of the work on radionuclide transport as a basis for identifying and selecting equations for food chain transport. However, the modeling of contaminant movement from soil to beef tissue or soil to dairy is not simple given the number of pathways by which cattle may come into contact with the contaminants originating in the floodplain. A high degree of uncertainty also is associated with the food chain transfer coefficients (discussed in the following section). Independent of the parameter uncertainty, and considering only the form of the models used, the uncertainty in the final risk estimates is likely to be affected by one to two orders of magnitude.

5.2.3.3 Parameter uncertainty

The equations used in estimating intake and dose are presented in Sect. 5.2.2. Each of these parameters or exposure factors is commonly treated as a single point estimate in human health risk assessment. In fact, none of these factors is truly a single value. This applies to exposure point concentrations, as well as component variables, such as contact rates, intermedia transfer factors, exposure frequency and duration, body weight, and surface area. The purpose of the parameter uncertainty analysis is to evaluate the uncertainty or variability surrounding the point estimates and the consequent effect on the integrity or reliability of the risk estimates.

It is important to distinguish between *variability* and *uncertainty* in risk assessment. Uncertainty refers to a lack of knowledge regarding the correct value for the given parameter or estimate. Variability in an exposure parameter reflects different levels of exposure experienced by different receptors in a population (EPA 1992d). An example of uncertainty would be the inability to select an appropriate BTF for a given chemical due to a lack of scientific information. An example of variability in exposure assessment is the range of values that may be appropriately used to characterize inadvertent soil ingestion for children. Sect. 5.5 provides a more detailed consideration of uncertainty versus variability in risk assessment.

Uncertainty analysis in the exposure assessment most often addresses the variability (i.e., as opposed to uncertainty, as defined above) surrounding the exposure parameters. The evaluation may be a qualitative or quantitative assessment. Difficulties in conducting a quantitative evaluation of uncertainty are noted by EPA in RAGS (EPA 1989a). A qualitative consideration is recommended by the Agency to provide order of magnitude indications of the influence of uncertainty/variability on the resultant risk estimates. However, a quantitative analysis of uncertainty, if it can be accomplished, is much more useful in revealing the limitations of the risk assessment. The baseline human health risk assessment of EFPC includes a quantitative evaluation of uncertainty.

EPA identifies several methods in conducting a quantitative analysis of uncertainty in exposure assessment: sensitivity analysis, analytical uncertainty propagation and use of classical statistical techniques, and probabilistic uncertainty analysis. The latter method (probabilistic uncertainty analysis) has been used in the BRA of EFPC. The risk assessment team has used Monte Carlo analysis simulation to characterize the exposure variables in the form of PDFs. The PDFs reflect the uncertainty/variability inherent in the exposure factors. Monte Carlo analysis is used to propagate this uncertainty/variability in the input variables (PDFs) and to generate a risk estimate in the form of a final probability distribution. These final distributions may then be statistically evaluated to generate an explicit risk estimate of known probability.

In the discussion below, PDFs are presented for the exposure factors used in the baseline human health risk assessment of EFPC. A discussion is provided outlining the basis for identifying or deriving the selected distribution for each exposure variable. Each probability distribution is then depicted graphically and a summary is provided to statistically characterize the PDF.

Four principal types of PDFs have been adopted in the BRA of EFPC to characterize the uncertainty/variability surrounding the exposure factors: a lognormal distribution, a truncated lognormal distribution, a truncated normal distribution, and a triangular distribution. The notation used to identify these PDFs is:

- **Lognormal:** $\text{Log}(m_{LOGX}, s_{LOGE})$

  where

  m_{LOGX} = mean of log base e,
  s_{LOGX} = standard deviation of log base e.

- **Truncated Lognormal:** $\text{TLog}(m_{LOGX}, s_{LOGX}, \min_{LOGX}, \max_{LOGX})$

  where

  Exp = exponential function,
  N = normal distribution,
  m_{LOGX} = mean of log base e,
  s_{LOGX} = standard deviation of log base e,
  $\min_{LOGX}$ = truncated minimum value of log base e,
  $\max_{LOGX}$ = truncated maximum value of log base e.

- **Truncated Normal:** N(m, s, min, max)

  where

  m = arithmetic mean of untransformed data,
  s = standard deviation of untransformed data,
  min = truncated minimum value of untransformed data,
  max = truncated maximum value of untransformed data

- **Triangular:** T(min, pk, max)

  where

  min = minimum value,
  pk = peak or most likely value,
  max = maximum value.

A triangular distribution is characterized by three parameters: the minimum of the distribution, the mode (or peak), and the maximum. The mode indicates the value most likely to occur, while the minimum and maximum specify the range of values that the variables assume. The following sections present the rationale behind the selection of PDFs for key exposure variables.

The PDFs for the exposure variables are summarized in Table 5.17 and described below. Figures of each of the PDFs are provided in Appendix O. The EFPC risk assessment team enlisted the assistance of Dr. David Burmaster and his staff at Alceon Corporation in identifying and selecting PDFs. Alceon Corporation, working as a subcontractor to SAIC, provided recommendations for PDFs for many of the exposure variables based upon Alceon's published work as well as work in progress. SAIC made use of these recommendations, modifying assumptions as needed to reflect site-specific considerations, and developed new PDFs as needed.

Section 5.1.3.1 presents a detailed discussion of data aggregation for the purposes of risk assessment. As noted, monitoring data for floodplain soils have been aggregated as a function of creek segment. In the deterministic risk assessment (Tier II evaluation) both the arithmetic mean and the 95% UCL on the arithmetic mean are used as exposure point concentrations. The mean and UCL values are derived by pooling all monitoring data for a given creek segment. All land use areas found within the creek segment are characterized by the same mean and UCL values (i.e., that are derived from the aggregated data set for the creek as a whole). However, for the purposes of the probabilistic risk assessment of EFPC (Tier III evaluation). The exposure point concentration must be treated as a PDF.

Table 5.17. Summary of distributions for baseline public health risk assessment input variables

Exposure Medium and Pathway	Distribution Type	Child	Adult	Reference
SURFACE WATER CURRENT AND FUTURE EXPOSURE				
Swimming (Dermal)				
Skin Area (cm^2)	Lognormal	9.14, 0.24	9.903, 0.096	EPA 1992b*
Skin Fraction (%)	Point Estimate	1.0	1.0	BPJ
Exposure Time (hr/d)				
Residential/Agricultural	Triangular	0.5, 2.6, 3.5	0.5, 2.6, 3.5	EPA 1992b*
Open Land Use	Triangular	0, 0.5, 2.6	0, 0.5, 2.6	EPA 1992b*
Exposure Frequency (d/y)				
Residential/Agricultural	Triangular	3, 7, 20	3, 7, 20	EPA 1992b*
Open Land Use	Triangular	0, 1, 7	0, 1, 7	EPA 1992b*
Body Weight (kg)	Lognormal	3.22, 0.34	4.35, 0.17	Brainard and Burmaster 1991
Wading (Dermal)				
Skin Area (cm^2)	Lognormal	9.14, 0.24	9.903, 0.096	EPA 1992b*
Skin Fraction (%)	Triangular	0.06, 0.21, 0.53	0.06, 0.21, 0.53	BPJ
Exposure Time (hr/d)				
Residential/Agricultural	Triangular	0.5, 1.3, 2.6	0.5, 1.3, 2.6	EPA 1992b*
Open Land Use	Triangular	0, 0.5, 2.6	0, 0.5, 2.6	EPA 1992b*
Exposure Frequency (d/y)				
Residential/Agricultural	Triangular	0, 7, 60	0, 7, 60	EPA 1992b*
Open Land Use	Triangular	0, 1, 7	0, 1, 7	EPA 1992b*
Body Weight (kg)	Lognormal	3.22, 0.34	4.35, 0.17	Brainard and Burmaster 1991
Ingestion (while swimming)				
Ingestion Rate (L/hr)	Triangular	0, 0.05, 0.075	0, 0.05, 0.075	EPA 1989a*, 1989b*
Exposure Time (hr/d)				
Residential/Agricultural	Triangular	0.5, 2.6, 3.5	0.5, 2.6, 3.5	EPA 1992b*
Open Land Use	Triangular	0, 0.5, 2.6	0, 0.5, 2.6	EPA 1992b*
Exposure Frequency (d/y)				
Residential/Agricultural	Triangular	3, 7, 20	3, 7, 20	EPA 1992b*
Open Land Use	Triangular	0, 1, 7	0, 1, 7	EPA 1992b*
Body Weight (kg)	Lognormal	3.22, 0.34	4.35, 0.17	Brainard and Burmaster 1991
SEDIMENT CURRENT AND FUTURE EXPOSURE				
Wading (Dermal)				
Skin Area (cm^2)	Lognormal	9.14, 0.24	9.903, 0.096	EPA 1992b*
Skin Fraction (%)	Triangular	0.06, 0.1, 0.47	0.06, 0.1, 0.47	BPJ
Adherence Factor (mg/cm^2-d)	Triangular	0.1, 0.3, 0.6	0.1, 0.3, 0.6	BPJ
Exposure Frequency (d/y)				
Residential/Agricultural	Triangular	0, 7, 60	0, 7, 60	BPJ
Open Land Use	Triangular	0, 1, 7	0, 1, 7	BPJ
Body Weight (kg)	Lognormal	3.22, 0.34	4.35, 0.17	Brainard and Burmaster 1991

* denotes BPJ based on the data provided in the reference.

Table 5.17. (continued)

Exposure Medium and Pathway	Distribution Type	Child	Adult	Reference
GROUNDWATER FUTURE EXPOSURE ONLY				
Ingestion (Tap-water)				
Ingestion Rate (L/d)				
Residential/Agricultural	Lognormal	-0.478, 0.5	0.115, 0.49	Roseberry & Burmaster 1991
Commercial	Lognormal	n/a	-0.5777, 0.49	EPA 1991a*
Exposure Frequency (d/y)				
Residential/Agricultural	Triangular	200, 350, 365	200, 350, 365	EPA 1991a*
Commercial	Triangular		200, 250, 300	EPA 1991a*
Body Weight (kg)	Lognormal	3.22, 0.34	4.35, 0.17	Brainard & Burmaster 1991
Inhalation of Vapors				
Inhalation Rate (m^3/hr)	Point	0.6	0.6	EPA 1989b*
Exposure Time (hr/d)	Lognormal	-2.16, 0.43	-2.16, 0.43	EPA 1989b*
Exposure Frequency (d/yr)	Triangular	200, 350, 365	200, 350, 365	EPA 1991a*
Body Weight (kg)	Lognormal	3.22, 0.34	4.35, 0.17	Brainard & Burmaster 1991
SOIL CURRENT AND FUTURE EXPOSURE				
Dermal Absorption				
Skin Area (cm^2)	Lognormal	9.14, 0.24	9.903, 0.096	EPA 1989b*
Skin Fraction (%)	Triangular	0.04, 0.1, 0.53	0.04, 0.1, 0.53	BPJ
Adherence Factor (mg/cm^2-d)	Triangular	0.2, 0.6, 1.2	0.2, 0.6, 1.2	BPJ
Exposure Frequency (d/y)				
Residential/Agricultural	Triangular	120, 270, 365	120, 270, 365	BPJ
Open Land Use	Triangular	5, 40, 200	5, 40, 200	BPJ
Body Weight (kg)	Lognormal	3.22, 0.34	4.35, 0.17	Brainard & Burmaster 1991
Ingestion				
Ingestion Rate (g/d)				
Residential/Agricultural	Lognormal	3.91, 1.15	3.69, 0.70	Calabrese et.al., 1989*
Open Land Use	Lognormal	3.69, 0.70	3.69, 0.70	EPA 1989*, 1991a*
Exposure Frequency (d/y)				
Residential/Agricultural	Triangular	120, 270, 365	120, 270, 365	BPJ
Open Land Use	Triangular	5, 40, 200	5, 40, 200	BPJ
Body Weight (kg)	Lognormal	3.22, 0.34	4.35, 0.17	Brainard & Burmaster 1991

* denotes BPJ based on the data provided in the reference.

Table 5.17. (continued)

Exposure Medium and Pathway	Distribution Type	Child	Adult	Reference
FOOD CURRENT AND FUTURE EXPOSURE				
Produce Ingestion				
Ingestion Rate (kg/d)	Lognormal	-2.41, 0.84	-2.18, 0.86	EPA 1989b[a], Yang & Nelson 1986[a]
Fraction from Area (%)	Triangular	0, 0.23[1], 0.4	0, 0.23, 0.4	EPA 1989a[a]
Exposure Frequency (d/y)	Triangular	100, 150, 365	100, 150, 365	BPJ
Body Weight (kg)	Lognormal	3.2, 0.34	4.35, 0.17	Brainard & Burmaster 1991
Meat Ingestion				
Ingestion Rate (kg/d)	Lognormal	-3.54, 0.73	-3.22, 0.72	Yang & Nelson 1986[a]
Fraction from Area (%)	Triangular	0, 0.44, 0.80	0, 0.44, 0.80	EPA 1989a[a]
Exposure Frequency (d/y)	Triangular	300, 350, 365	300, 350, 365	BPJ
Body Weight (kg)	Lognormal	3.2, 0.34	4.35, 0.17	Brainard & Burmaster 1991
Fish Ingestion				
Ingestion Rate (kg/d)	Lognormal	-4.25, 0.84	-3.77, 0.67	EPA 1989b[a]
Fraction from Area (%)	Triangular	0, 0.01, 0.07	0, 0.01, 0.07	EPA 1989a[a], BPJ
Exposure Frequency (d/y)	Triangular	300, 350, 365	300, 350, 365	BPJ
Body Weight (kg)	Lognormal	3.22, 0.34	4.35, 0.17	Brainard & Burmaster 1991
FOOD FUTURE EXPOSURE ONLY				
Milk/Dairy Ingestion				
Ingestion Rate (kg/d)	Lognormal	-1.36, 0.68	-1.83, 0.72	Yang & Nelson 1986[a]
Fraction from Area (%)	Triangular	0, 0.44, 0.80	0, 0.44, 0.80	EPA 1989a[a]
Exposure Frequency (d/y)	Triangular	200, 350, 365	200, 350, 365	BPJ
Body Weight (kg)	Lognormal	3.22, 0.34	4.35, 0.17	Brainard & Burmaster 1991

[a] denotes BPJ based on the data provided in the reference.

The PDF for all exposure point concentrations (EPCs) is characterized as a truncated normal distribution of the following form:

EPC: TN(m, s, min, max)

Note that a normal distribution is used for exposure point concentration term regardless of the underlying shape of the distribution of the monitoring data. Environmental quality data are typically lognormally distributed. The EFPC risk assessment team believes, however, that a normal distribution is always appropriate. (The reader is referred to Sect. 5.1.3.3 for further clarification.) Essentially, a normal distribution is used as the basis of the PDF because we are working with a *distribution of mean values* (i.e., distribution of arithmetic means). By the Central Limit Theorem, the distribution of means is always normally distributed regardless of the shape of the distribution from which samples are obtained.

In order to gain confidence that the PDF for exposure point concentration meaningfully characterizes the range of possible average daily intake/dose estimates, an additional evaluation was conducted. Exposure point concentrations (mean and UCL values) were determined based on data aggregated by *land use area* rather than *creek segment*. Aggregation by land use area was not initially conducted because of the limited data set available as a function of land use area. EFPC is a very large study area, and even though thousands of samples have been obtained, the data density as a function of land use area is often inadequate. Nevertheless, mean and UCL values for the creek segments as a whole are compared with the mean and UCL values for each land use area found within a given segment. These results are presented in Appendix M.

The tables in Appendix M express the mean and UCL concentration for each land use area as a percentage of the mean and UCL values for the creek segment (within which the land use areas are contained). The chemicals included are the inorganic elements measured by NAA techniques. The results of the comparison generally do not indicate great variations in summary statistics based on land use aggregation versus creek segment aggregation. To ensure that the variation in exposure point concentration was being incorporated in the probabilistic risk assessment, the UCL values for each COC in each land use segment were compared to the PDF derived by Monte Carlo simulation. The EFPC risk assessment team needed to confirm that the highest UCLs determined for a given land use area would fall within the exposure point concentration PDF derived for the corresponding creek segment as a whole. In all cases, this was shown to be the case.

It is concluded that the PDFs for exposure point concentration of chemicals in floodplain soils meaningfully characterize (encompass) the uncertainty in the mean and UCL point estimates. The truncated normal distribution may be appropriately used.

Body Weight Distribution for Adults and Children. Data on body weight distributions are available for adults, but not for children. EPA recommends use of a point estimate of 70 kg for adult body weight when conducting a human health risk assessment. However, the available data on body weight are of high quality, allowing a PDF to be established for use in risk assessment. The data for adult body weights are found in Brainard and Burmaster (1991). The body weight of adult men is well-represented by a single lognormal distribution. Based on this study, the following distribution has been selected to represent the body weight distribution of adults (measured in kg) for all exposure pathways examined at EFPC:

$$BW_a: \text{Log}(4.35, 0.17)$$

This distribution was developed based on data for men only and corresponds to a mean of 77.5 kg; however, its use for men and women will have little effect on the final result.

No analytical data are available to derive body weight distributions of children ages 3 to 12. However, data on weight percentiles for each year of age were presented in NHS (1987) and used to establish a PDF to portray the overall distribution of weight in the full age range 3 through 12 years. Since the weights of boys and girls in this age range do not differ appreciably, the analysis was conducted for boys only and the resulting distribution is used for girls as well.

The natural logarithm of the weight for each percentile of each 1-year age group was plotted on probability paper. These plots confirmed a lognormal distribution for the weight in each of the nine age groups. The standard deviations were determined for each age group, and are shown along with the median values in Table 5.18. Using these distributions, a Monte Carlo simulation was conducted by drawing 100 ranged samples from each age group (Crystal Ball™, Decisioneering 1991). The resulting 900 samples were then pooled and used as the sample population. The points on the probability plot of the 900 samples form a straight line except at the ends (outside $\pm$ two standard deviations from the mean), indicating that it is appropriate to use a lognormal distribution to represent weight. The parameters of the distribution of childrens' body weight, measured in kg, are:

$$BW_c: \text{Log}(3.22, 0.34)$$

Table 5.18. Parameters of the lognormal distributions for body weight (kg) for age groups 3 through 12 years

Age Group	Log Mean	Log Standard Deviation
$3 \leq$ age < 4	2.75	0.114
$4 \leq$ age < 5	2.87	0.133
$5 \leq$ age < 6	2.99	0.138
$6 \leq$ age < 7	3.13	0.145
$7 \leq$ age < 8	3.21	0.151
$8 \leq$ age < 9	3.33	0.181
$9 \leq$ age < 10	3.43	0.165
$10 \leq$ age < 11	3.59	0.195
$11 \leq$ age < 12	3.69	0.252

This distribution will be used for all children aged 3 through 12 years with a corresponding mean of 25.0 kg.

Total Body Surface Area. The determination of dermal exposure requires a knowledge of total body surface area as well as the fraction of skin exposed. Information on skin fraction for the dermal exposure pathways is provided below.

Point estimates of surface area for male and female adults and different age groups of children have been used in a variety of studies on dermal exposure (EPA 1989c, 1989b). For example, the commonly accepted 50th percentile for total body surface area is 19,400 cm^2 for males and 16,900 cm^2 for females. These values may be used in calculating a simple average estimate of dermal exposure. However, the available data are of sufficient quality to establish a PDF of surface area for use in risk assessment. This section describes the methods used to derive distributions of total body surface area for adults and children.

EPA (1989c) conducted a thorough search of the literature regarding the surface area of the human body. Researchers used a variety of measurement techniques to formulate a relationship between surface area, and body height and weight. The most widely accepted model for this relationship is:

$$SA = a\, H^b\, W^c \qquad (25)$$

where

SA = surface area (m^2),
H = height (cm),
W = weight (kg),
a,b,c = parameters estimated on the basis of measurements.

Dubois and Dubois (1916) originally proposed this model and many other researchers have used it and refined the estimation of the parameters (Boyd 1935, Gehan and George 1970, EPA 1989b). The research by Gehan and George (1970) involved the analysis of measurements of 401 individuals, by far the largest number up to that time. EPA used the same 401 data observations and performed a regression analysis, which resulted in minor revisions to the parameters. These parameters are the most accurate and have been used in this assessment.

Costeff (1966) developed a different model, which is used to estimate surface area on the basis of weight only. This formula is:

$$SA = \frac{4W+7}{W+90} \qquad (26)$$

where

SA = surface area (m^2),
W = weight (kg).

This model originally was intended for use by physicians in need of a simple bedside formula to calculate skin area in order to prescribe medication. However, using the parameters established by EPA, this formula has been shown to be surprisingly accurate in comparison to the Dubois and Dubois (1916) model, and is recommended for use in situations where data on height are unavailable.

To develop a PDF for adults, the surface area formula based on the parameters refined by EPA was applied to a bivariate distribution for height and weight of men (Brainard and Burmaster 1991). A Monte Carlo simulation was conducted by drawing 5000 random samples from the bivariate distribution and the resulting surface area was calculated (Crystal Ball™, Decisioneering 1991). The median of the surface area distribution is 19,600 cm^2, a value that is remarkably close to the point estimate that has been used by EPA.

A probability plot of the natural logarithm of surface area for the 5000 samples was created using Systat (1990). The points form an almost straight line, indicating that surface area can be well-represented by a lognormal distribution. A regression analysis was performed using Mathmatica™ 2.0 (Wolfram 1991) and the R^2 value was determined to be 0.9998. The parameters of the distribution, with the surface area measured in cm^2, are:

$$SA_a: \text{Log}(9.903, 0.096)$$

This distribution is used for males and females. It is believed that this will not have a noticeable effect on the outcome of the analysis, while developing an average of male and female values would be undesirable.

Since bivariate data for height and weight were available for children aged 3 through 12, a different method was used to determine a PDF for the surface area of children. The lognormal distribution for weight of boys was used to calculate the surface area based on the Costeff (1966) formula. A Monte Carlo simulation was conducted by drawing 5000 random samples from the weight distribution, and the resulting surface area was calculated. The median of the surface area distribution is 9300 cm^2.

A probability plot for the natural logarithm of surface area (SYSTAT 1990) showed that the points form a reasonably straight line, indicating a lognormal distribution. The parameters of this distribution of surface areas for children, measured in cm^2, are:

$$SA_c: \text{Log}(9.14, 0.24)$$

While this distribution is based only on data for boys, weights for boys and girls in the age range of interest differ only slightly, and consequently, this distribution has been used for girls as well as boys.

Skin Fraction for Dermal Exposure Pathways. Widely accepted data are available on the percent of total skin area for individual parts of the human body (EPA 1984c, 1989b). However, data are lacking on the fraction of the body exposed during various activities, such as swimming, wading, and everyday life. The PDF derived for this variable is based on guidance from EPA (1989a) and professional judgment regarding recreational activities along EFPC. A point estimate has been used to estimate the skin fraction exposed during swimming, and this parameter is not characterized by a PDF for the swimming pathway only. This is because the whole body is assumed to be covered with water at some point during this kind of activity.

A triangular PDF is appropriate for use in the absence of a large data set. Available data are insufficient to justify the use of a normal or lognormal distribution. Use of a uniform distribution is inappropriate, since it would imply that all values within a given range are equally likely to occur. This is clearly not the case for wading, during which a person is probably most likely to get water on the feet, lower legs, and hands, and also less likely to include the upper leg or, alternatively, to only wet the feet and ankles. Therefore, a triangular PDF will be used to describe the fraction of skin area exposed during wading and recreational activities.

The following information indicates the minimum, mode, and maximum body parts that are considered to be exposed, and their corresponding fraction of the total surface area for each pathway. For all of the dermal exposure pathways, the fractions are considered to be the same for residents, homesteaders, and individuals exposed under the open land use scenario.

Exposure Pathway: Dermal Contact with Water During Wading

Minimum: Feet
Mode: Feet, lower legs, and hands
Maximum: Lower extremities, upper and lower arms, and hands
Distribution: T(0.06, 0.21, 0.53).

Exposure Pathway: Dermal Contact with Sediment Water During Wading

Minimum: Feet
Mode: Feet and hands
Maximum: Feet, lower legs, and hands
Distribution: T(0.06, 0.10, 0.47).

Exposure Pathway: Dermal Exposure to Contaminated Soil

Minimum: Hands
Mode: Feet and hands
Maximum: Lower extremities, upper arms, forearms, hands
Distribution: T(0.04, 0.10, 0.53).

Surface Water Exposure (While Swimming). Incidental ingestion of surface water may occur during swimming. The point estimate for the incidental ingestion rate during swimming is based on the value of 0.05 L/hr that is presented in EPA (1989a) and used for the MLE and RME assumptions for all age groups in the exposure assessment. Because the data used to derive this value are not of high quality, the incidental ingestion rate is best represented by a triangular distribution with the point estimate used to represent the mode. The minimum and maximum

values are based on professional judgment. Therefore, the triangular distribution is characterized by the following values (in L/hr):

$$IR_{SW} = T(0, 0.05, 0.075)$$

Point estimates of exposure frequency while swimming and wading were based on national recreational swimming frequency data recommended by EPA (1988c; 1989a,b) and on professional judgment. Because it is not possible to use the data to determine the shape of the PDF, exposure frequency is represented by a triangular distribution.

For the residential and agricultural exposure scenarios, the shape of the triangular distribution for swimming exposure frequency uses the MLE value of 7 days per year to represent the mode. The minimum and maximum values are based on professional judgment. The maximum value of the distribution was selected so the recommended RME point estimate fell between the 90th and 95th percentile of the distribution. Therefore, for the residential and agricultural scenarios, swimming frequency is characterized by the following distribution:

$$EF_{SW,R/A}: \ T(3, 7, 20)$$

A triangular distribution also is used to describe swimming frequency for individuals under the open land use scenario. In the absence of site-specific information, the point estimate value for the MLE is used to estimate the mode, while the national average value of 7 days per year is used to represent the maximum value. The minimum exposure frequency value is set at zero, in order to include those individuals who do not swim in EFPC. Therefore, the PDF for swimming frequency is:

$$EF_{SW/WAD,O}: \ T(0, 1, 7)$$

For the residential and agricultural scenarios, wading is assumed to occur with much lower frequency than swimming. Because of the professional judgment that was required to develop the point estimates for wading frequency, a triangular distribution is used to describe this parameter. The open land use scenario uses the same exposure frequency and PDF for wading as for swimming.

For the residential and agricultural scenarios, the mode of the distribution is equal to the MLE point estimate and the maximum value is established so that the RME point estimate falls at approximately the 90th to 95th percentile. Maximum and minimum values are based on professional judgment, resulting in the following distribution:

$$EF_{WAD,R/A}: \ T(0, 7, 60)$$

The point estimates for swimming and wading exposure time were based on a data set that cannot be used to determine the shape of a PDF. Therefore, triangular distributions are used to describe the PDFs for swimming and wading exposure time.

For the residential and agricultural scenarios, the distribution of exposure times for swimming are based on the MLE and RME point estimates. The minimum value on the distribution is equal to 0.5 hr, which is the exposure time recommended by EPA (1992b) for this activity. The RME point estimate value of 2.6 hr is used to describe the mode of the distribution, while the maximum value is set at 3.5 hr. This last value is based on professional judgment. Therefore, the distribution for swimming exposure time for the residential and agricultural scenarios is:

$$ET_{SW,R/A}: \ T(0.5, 2.6, 3.5)$$

For the open land use scenario, the triangular distribution for swimming exposure time is characterized by a mode of 0.5 hr based on the EPA (1992b) recommendation. The maximum value is 2.6 hr, which is the RME point estimate, and the minimum value is set at zero to capture those individuals who do not swim in EFPC. Thus, the distribution is:

$$ET_{SW,O}: \ T(0, 0.5, 2.6)$$

The distributions for wading time also are characterized by triangular distributions. It is assumed that the time devoted to wading is generally shorter than the time devoted to swimming. For the residential and agricultural scenarios, the distribution is described by a maximum value of 2.6 hr, and a minimum value of 0.5 hr. The basis for these values is described above. Based on professional judgment, the mode of the distribution is set at one-half the maximum value (i.e., 1.3 hr). Therefore, the wading time distribution is:

$$ET_{WAD,R/A}: \ T(0.55, 1.3, 2.6)$$

For the open land use scenario, wading is assumed to occur with the same exposure time as for swimming, and is characterized by the same PDF:

$$ET_{WAD,O}: \ T(0, 0.5, 2.6)$$

A triangular distribution is used to characterize the PDFs for the adherence factor because site-specific data are not available on adherence of EFPC soil or sediments to human skin, and there is a lack of data on which to base the recommended values for soil adherence.

For the soil to skin adherence factor PDF, the MLE value of 0.6 mg/cm^2 recommended by EPA (1991c) is used to estimate the mode, and the recommended value of 0.2 (EPA 1992b) is used to estimate the minimum value of the distribution. The maximum value is set at twice the mode. This is based on professional judgment. Therefore, the triangular distribution for soil adherence (in mg/cm^2) is:

$$AF_{SOIL}: \ T(0.2, 0.6, 1.2)$$

For the sediment to skin adherence factor, EPA indicates that adherence factors for some sediments (particular sandy sediments) are likely to be "much less" than for soils because contact with water may wash the sediment off the skin (EPA 1989a). In the absence of data, the values of the distribution for the sediment to skin adherence factor are set equal to one-half the values used in the distribution for soils exposure. Therefore, the distribution for the sediment to skin adherence factor is characterized by the following triangular distribution:

$$AF_{SED}: \ T(0.1, 0.3, 0.6)$$

Groundwater Ingestion Rate and Exposure Frequency. To characterize the risk resulting from the ingestion of contaminated drinking water, it is necessary to assess the rate at which tap water is ingested and the frequency of exposure to the water. This section explains the distributions for the two variables.

EPA and other agencies have used a point estimate for water ingestion of 1 and 2 liters of water per day for children and adults, respectively (EPA 1989a,b). More recently, Roseberry and Burmaster (1991) reported distributions for total water intake and tap water intake by age group. Total water intake includes water from other sources (e.g., water contained in food and bottled beverages). The median tap water intakes reported by Roseberry and Burmaster (1991) are 1.122 L/day for adults and 0.620 L/day for children aged 1 through 10. Roseberry and Burmaster (1991) found that the distributions were well-represented by lognormal distributions. On the basis of their findings, the following lognormal distributions, with water ingestion rate measured in L/day, will be used for residents and homesteaders:

$$IR_{DW,A}: \ Log(0.1151, 0.49)$$
$$IR_{DW,C}: \ Log(-0.478, 0.50)$$

For commercial receptors, it will be assumed that one-half of daily water intake occurs at work. This EPA (1991a) recommendation will be used for calculating drinking water exposure to a

commercial receptor. By maintaining the shape of the distribution, the following lognormal distribution will be used for adults under the commercial exposure scenario:

$$IR_{DW,\ COMM}: \text{Log}(-0.5777, 0.49)$$

Distributions for exposure frequency are based on professional judgment as well as the RME value of 350 days per year for residents and 250 days per year for commercial adults recommended by EPA (1991a). Because of the lack of data, exposure frequency is represented best by a triangular distribution, with the MLE value as the peak (or mode) of the triangle. The following triangular distributions will be used, with exposure frequency measured in days per year:

$$EF_{DW,\ A/R}: \text{T}(300, 350, 365)$$
$$EF_{DW,\ COMM}: \text{T}(200, 250, 300)$$

Inhalation Exposure: Vapors While Showering and Dust/Particulates while Mowing. Inhalation of organic vapors released from tap water while showering and inhalation of dust and particulates released during mowing of hay by homesteaders are two pathways of concern. This section explains the distributions for inhalation rates, exposure time, and exposure frequency, the three variables of interest for the inhalation pathways.

EPA (1989b) has presented data on inhalation rates for various physical activities. However, these data are not of high quality, and because of the uncertainty associated with such estimates, fixed point estimates for inhalation rates will be used instead of PDFs to calculate inhalation exposure for both exposure pathways.

Data on shower duration reported by EPA (1989b) were used to determine distributions of exposure time while showering. Percentile values for shower duration were plotted on probability plot graph paper, and the data points appear as a straight line above the 3rd percentile and below the 99th percentile, suggesting that the data are lognormally distributed. The PDF for shower exposure time, expressed in fraction of an hour, is:

$$ET_{SHOW}: \text{Log}(-2.16, 0.43)$$

This distribution will be used for both adults and children because insufficient data are available on bathing times for children.

The distribution for inhalation exposure time for the mowing pathway is based on professional judgment. Given the lack of data, a PDF will not be developed for this parameter. A point estimate will be used to model exposure time for the mowing pathway.

A triangular distribution will be used for exposure frequency for all inhalation pathways because of the nature of the variable and the lack of data. The distributions are based on professional judgment with the use of some basic data from EPA (1989b, 1991a). The vapor inhalation pathway for residents and homesteaders, children and adults, have obvious maximum exposures of 365 days per year for those who never leave the EFPC area. The RME value of 350 days per year is appropriate for the mode of the distributions. For showering, this number has been suggested by EPA (1989b), but may exceed the actual exposure frequency, since most people are expected to be away from their homes for approximately 2 weeks per year. A minimum of 300 days per year provides a lower bound for those who are out of the study area 1 day per week in addition to a 2-week absence. Therefore, the exposure frequency distribution is 300 days per year for the minimum, 350 days per year for the most likely, and 365 days per year for the maximum. This is expressed as:

$$EF_{SHOW}: \; T(300, 350, 365_x)$$

For the mowing pathway, a lack of data leads to the conclusion that exposure frequency is best estimated using a point estimate rather than a PDF.

Ingestion Rate and Exposure Frequency for Contaminated Soil. Inadvertent ingestion of contaminated soil is a pathway of concern at EFPC. To characterize this exposure pathway, it is necessary to assess the rate at which soil is ingested and the frequency of exposure to the soil. This section explains the distributions for the two variables.

Point estimates of soil ingestion rates are based on data reported in several documents (Calabrese et al. 1989; EPA 1989b, 1991a). For adults under three exposure scenarios (i.e., residential, homesteader, and open land use) the MLE is 40 mg/d and the RME is 100 mg/d. For children under the residential or homesteader scenarios, the MLE and RME values are based on a weighted average for the ages 3 through 12 years, and are 50 mg/d and 133 mg/d, respectively. For the open land use scenario, the RME and MLE for children are based on a weighted average of ingestion rates for ages 9 through 18 and are similar to the adult ingestion rates: the MLE is 40 mg/d and the RME is 100 mg/d.

The MLE and RME ingestion rates were used to determine the shape of the distributions. Thompson and Burmaster (1991) have demonstrated that soil ingestion rates for children are

lognormally distributed. This assumption is assumed to also be true for adults. The MLE is interpreted as the 50th percentile and the RME as the 90th percentile. The natural logarithm of these ingestion rate percentiles was calculated and the results for children and adults were plotted separately on probability plot graph paper. The standard deviation was calculated from the slope of each line. The following lognormal distributions were obtained for soil ingestion rates (measured in mg/d):

$$IR_{SOIL,A}: \text{Log}(3.69, 0.70)$$
$$IR_{SOIL,C}: \text{Log}(3.91, 1.15)$$

These are equivalent to a mean and standard deviation of 40 $\pm$ 2.01 mg/d for adults and 50 $\pm$ 3.16 mg/d for children.

Monte Carlo simulation was used to further explore the character of this distribution. From a statistical analysis of the PDF generated, it was determined that the outlying estimate of soil intake obtained from this distribution (i.e., at probabilities greater than 90th percentile estimates) were unrealistically high. Therefore, a truncated lognormal distribution was selected as the PDF for the soil ingestion variable (units mg/d):

$$IR_{SOIL,A}: \text{TLog}(3.69, 0.70, 0, 5.298)$$
$$IR_{SOIL,C}: \text{TLog}(3.91, 1.15, 0, 5.704)$$

The four numbers provided for each of the distributions above are the log-scale mean, standard deviation, the lower truncation limit, and the upper truncation limit. These correspond to 40, 2.01, 1, and 200 mg/d for adults, and 50, 3.16, 1, and 300 mg/d for children on the untransformed scale.

The distribution of exposure frequency is based on the MLE and RME values. Because of a lack of data, these values are based on professional judgment, and are best represented by a triangular distribution with adults, children, residents, and homesteaders being treated identically. A mode of 270 days per year that results from a daily exposure for 9 months out of the year is reasonable. A maximum exposure of 350 days per year allows for a daily exposure, with approximately 2 weeks of absence from the EFPC area. A minimum of 120 days per year indicates a minimum exposure frequency for an individual who is exposed only 5 days per week for 6 months of the year. Thus, the triangular distribution of exposure frequency for adults and children, residents and homesteaders, measured in days per year is:

$$EF_{SOIL,R/A}: \text{T}(120, 270, 350)$$

For the open land use scenario, a mode of 40 days per year describes occasional recreational users of the contaminated area; a maximum of 200 days per year describes schoolchildren trespassing across the contaminated area on their way to school. A minimum of 5 days per year describes an occasional use of the area. The triangular distribution to describe exposure frequency under the open land use scenario is:

$$EF_{soil,o}: T(5, 40, 200)$$

Food Chain Pathways: PDFs for Ingestion Rates, Fraction Ingested from Contaminated Area, and Exposure Frequency. The consumption of agricultural products originating from EFPC may present health risks as a result of the ingestion of contaminants that are incorporated into those products. Food chain exposure pathways of concern include ingestion of produce that has been grown on contaminated soil, the consumption of beef or milk from cattle grazing on contaminated forage, and the consumption of fish caught from EFPC. A number of parameters must be quantified to characterize the risks associated with ingestion exposure. These parameters are the concentration of chemicals in the food product, the quantity of each food type ingested, the fraction of that food item that originates from the contaminated area, and the frequency of exposure. This section describes the methods used to estimate distributions and the variables needed to calculate exposure through the food chain.

Different methods were used to determine the shape and characteristics of the ingestion rate distributions. The EFPC risk assessment team reviewed the published literature for studies that would be relevant to this effort. In addition, the team statistically evaluated the available data to derive PDFs when possible. For produce and fish ingestion rates, the shape of the distributions were determined by evaluating existing data. For meat and milk ingestion rates, the shape of the distributions were assumed based on previously published information.

The values of the parameters that describe the lognormal distributions (i.e., log mean and log standard deviation) were determined differently for each of the food groups. The parameters used to describe the produce ingestion rate PDF were determined from the values of the point estimates (i.e., the RME and MLE) used in the Tier II deterministic risk assessment. The parameters of the PDFs for meat and milk ingestion were determined based on published survey results (Yang and Nelson 1986). The parameters for the fish ingestion rate PDF were determined from the slope and intercepts of probability plots/regression analyses generated using survey data. The choice of methods for determining the values of the distribution parameters was principally based on an assessment of the quality of the available data. These methods are described in detail below.

Produce ingestion rates based on survey results were reported in EPA (1989b) and Yang and Nelson (1986). The data presented in EPA (1989b) included the percentiles for ingestion rates of different produce groups, but not separate ingestion rates for adults and children. The percentiles of the ingestion rates reported were used to determine the shape of the distribution. The natural logarithm of the ingestion rate percentiles were plotted on probability plot graph paper, and the percentile points were found in an almost straight line, indicating a lognormal distribution. This agrees with the findings of McKone and Ryan (1989) based on their evaluation of the ingestion data presented in Yang and Nelson (1986).

The parameters that describe the lognormal distribution (i.e., m and s) were estimated from the point estimates of produce ingestion rates for adults and children. The derivation of the point estimates for produce ingestion rates were based on data presented in EPA (1989b) and Yang and Nelson (1986) and are described in detail in Sect. 5.2.2.10. For adults and children, the lognormal mean was calculated from the natural logarithm of the MLE point estimate. The standard deviation was calculated from the RME and MLE point estimates using the following formula (Aitchison and Brown 1976):

$$Log(s) = \frac{Log(RME) - Log(MLE)}{1.28} \qquad (27)$$

where

s = standard deviation,
1.28 = the 90th percentile of the standard normal distribution.

The distributions of total daily ingestion of produce, with ingestion rates measured in kg/day, are expressed as follows:

$IR_{PROD,A}$: Log(-2.18, 0.86)
$IR_{PROD,C}$: Log(-2.41, 0.84)

A lognormal distribution is used to describe the ingestion rates of meat and milk. The assumption of lognormality is based on professional judgment and supported by evidence from McKone and Ryan (1989), who assumed that all food ingestion rates, including meat and milk ingestion rates, were lognormally distributed. In addition, the standard deviations of the data for milk and meat ingestion rates presented by Yang and Nelson (1986) were typically more than the mean values. This suggests that a skewed distribution such as a lognormal distribution is appropriate.

The parameters for the lognormal distributions were derived from milk and meat ingestion rate data presented by Yang and Nelson (1986). These data summarize consumption rates for more than 30,000 individuals in 114 primary sampling units. The mean values for consumption rates are the means of the means for the 114 sampling units, and the standard error value is the standard deviation of the sample means among the 114 sampling units (McKone and Ryan 1989). The PDF must be determined based on the distribution of intake among individuals, and the upper bound must correspond to the upper bound individual intake, rather than the upper bound for the population intake. Sample sizes for each age group were determined using the total sample size and the percentage values for the different age groups that made up the sample. These sample sizes were used to calculate the standard deviation as follows (Aitchison and Brown 1976):

$$s = \sqrt{n}\ x\ s.e. \tag{28}$$

where

s = standard deviation on the original scale,
n = sample size of each age category,
s.e. = standard error around the mean.

Yang and Nelson (1986) presented results by age category. To derive an overall distribution for adults, the age-specific means and variances were averaged over categories, weighted by the population size within each category (age groups 20-24, 25-29, 30-39, and 40-59). The means and standard deviation were used to calculate the parameters of the lognormal distribution. First, the log-transformed standard deviation (s_{LOGX}) was calculated:

$$s_{LOGX} = \sqrt{Ln\left(\frac{s_x^2}{m_x^2} + 1\right)} \tag{29}$$

then it was used to calculate the log mean:

$$m_{LOGX} = Log(m_x) - 0.5\ x\ (s_{LOGX})^2 \tag{30}$$

The lognormal distribution for beef ingestion, with ingestion rates in units of kg/day, is:

$$IR_{BEEF,\ A}: \text{Log}(-3.22, 0.72)$$
$$IR_{BEEF,\ C}: \text{Log}(-3.54, 0.73)$$

The lognormal distribution for milk ingestion, with ingestion rates represented in units of kg/day, is:

$$IR_{MILK,\ A}: \text{Log}(-1.83, 0.72)$$
$$IR_{MILK,\ C}: \text{Log}(-1.36, 0.68)$$

The shape of the distribution for fish ingestion was determined from data presented by EPA (1989b). The percentiles for fish ingestion based on the data for total consumption of fish and shellfish by sport fishermen in the Metropolitan Los Angeles area (EPA 1989b) were plotted on probability plot graph paper. The points formed an almost straight line on the graph, indicating a lognormal distribution to the fish ingestion rate.

The parameters that describe the distributions for fish ingestion rates for children and adults were based on the MLE and RME point estimate values. Point estimates for fish ingestion rates were determined from the data reported by Yang and Nelson (1986) and EPA (1989b and 1991a) and are described in Sect. 5.2.2.11.

To determine the parameters that describe the distribution, the natural logarithm of the MLE and RME values were plotted separately for adults and children on probability plot graph paper, and the slopes of the lines were used to estimate the standard deviations of the distributions. The lognormal mean was determined by the intercept. The distributions for total daily ingestion of fish, measured in kg/day, are as follows:

$$IR_{FISH,\ A}: \text{Log}(-3.77, 0.67)$$
$$IR_{FISH,\ C}: \text{Log}(-4.25, 0.84)$$

A triangular distribution was used to describe the fraction of homegrown produce, meat, or recreationally caught fish that originate from the contaminated area. A triangular distribution is appropriate where data that can be used to describe a distribution are lacking.

The distribution for the fraction of produce ingested from the contaminated area is based on an MLE of 0.23 and an RME of 0.35 derived from data on consumption of homegrown fruits and vegetables (EPA 1989b). A triangular distribution was developed based on these values,

where the numbers indicate the minimum, mode, and maximum for distribution of fraction ingested:

$$FI_{PROD}: \ T(0.0, 0.23, 0.40)$$

The distribution for the fraction of meat and milk ingested that originates from the contaminated area is based on an MLE of 0.40 and an RME of 0.75 derived from EPA (1989a,b). A triangular distribution was developed based on these values for use in the beef and milk ingestion calculations:

$$FI_{BEEF/MILK}: \ T(0.0, 0.44, 0.80)$$

The distribution for the fraction of fish ingested from the contaminated area is based on professional judgment. Because of the lack of data, a triangular distribution for fraction ingested is used. The values of the point estimates for the fraction ingested were 0.01 for the MLE and 0.05 for the RME. These values were used to develop the following triangular distribution:

$$FI_{FISH}: \ T(0, 0.01, 0.07)$$

PDFs describing exposure frequency for produce, meat, milk, and fish ingestion are based on professional judgment and the MLE and RME point estimate values. Because of the lack of data, exposure frequency is best represented by a triangular distribution, with the MLE of 150 days/year as the peak (or mode) of the triangle. The PDF describes exposure frequency to both residents and homesteaders for produce ingestion and fish ingestion. Only homesteaders are assumed to ingest home-produced beef and milk. The following triangular distributions are used, with exposure frequency measured in days per year:

$$EF_{PROD}: \ T(100,150,365)$$
$$EF_{BEEF/MILK/FISH}: \ T(200,350,365)$$

Chemical-specific BTFs are used to calculate chemical concentrations in produce grown on contaminated soil.

Point estimates for soil to plant B*v*s for inorganic chemicals have appeared in several publications (EPA 1985c, 1990d; Belcher and Travis 1989; Baes et al. 1984; Hoffman and Baes 1979; Ng 1982; Ng et al. 1977; NCRP 1989, 1991). Point estimates for feed-to-milk and feed-to-meat biotransfer factors (B*m*s and B*a*s, respectively) for inorganic chemicals are presented

in Belcher and Travis (1989), NCRP (1989, 1991), and Baes et al. (1984). These BTFs are largely based on data collected by Ng (1982) and Ng et al. (1977).

A literature search and review was undertaken in an attempt to independently characterize the range of BTF values used for the food chain exposure pathway at EFPC. Data obtained from the EPA (1985c) series, individual peer-reviewed documents, and reviews were tabulated, and where necessary, converted to consistent units.

The transfer factors provided by NCRP (1991) for soil to plant biotransfer were not encompassed by the distributions determined by Belcher and Travis (1989). For some chemicals, such as mercury, the BTF from the NCRP publication was found to exceed the 100th percentile on the Belcher and Travis distribution; whereas for others elements, such as beryllium, the NCRP value was unreasonably low on the distribution. Because of these discrepancies, and because the distributions provided by Belcher and Travis were based on a limited number of data points, a literature search was undertaken to determine the distribution of B_v for selected chemicals: arsenic, cadmium, mercury, beryllium, and manganese. A data-set was created based on a combination of literature on bioaccumulation, sludge application literature, the SAIC field study data, and the values reported by Baes et al. (1984) and NCRP (1991). This data set was, therefore, established with the intention of including as much of the useful information as possible. Tables M.4 through M.8 in Appendix M provide a listing of the B_vs determined for each chemical and plant, along with the associated reference.

Use of conversion factors was sometimes necessary to convert transfer factors obtained from the different literature sources into consistent units. Sludge application studies often report plant concentrations, sludge concentrations, and sludge application rates. In a few cases, B_vs were reported in units of (mg chemical/kg plant)/(kg chemical/ha). Application rates were converted to soil concentrations using the element concentrations in the sludge, and assuming a soil density of 2,000 metric tons per hectare (calculated as 1.33 g/cm^3) for a plow depth of 15 cm. This is similar to the EPA (1990c) default soil dry mass of 2.7×10^9 g/ha (calculated as 1.35 g/cm^3) for a 20 cm plow depth.

The SAIC field study and other studies reported B_v or chemical concentrations in plants in terms of wet weight only. Other studies reported data in terms of dry weight. All transfer factors were converted to wet weight units using the default water contents of the associated plant parts shown in Table 5.9.

Insufficient data are available in the literature on soil-to-plant transfer factors expressed as a function of plant type. It is difficult, therefore, to develop PDFs by plant type, for use in

Monte Carlo simulation. Given this limitation, the EFPC risk assessment team derived new PDFs based on an expanded, pooled data set. For each chemical under evaluation at EFPC, the team aggregated available data on B_v values (point estimates): leafy vegetables, roots, grains, garden fruits, and legumes. A probability density function was derived for the B_v parameter, for a given chemical, based on this larger data set. A series of probability plots (P-Plot) were prepared using SYSTAT (SYSTAT 1990) and a regression line was fit to the data. A good fit was observed following log-transformation of the data. A lognormal distribution was, therefore, assumed as the shape of the PDF for the B_v parameter. The selection of a lognormal distribution is supported by the work of Belcher and Travis (1989), McKone and Ryan (1989), and Ng (1982). Mean and standard deviations (in log units) were calculated from the regression data and used to define the PDF.

The PDFs for the B_v parameter derived by the EFPC risk assessment team were compared with the distributions published by Belcher and Travis (1989). The PDFs derived for the EFPC study are found to be broader and more inclusive. The B_v values published in NCRP (1991) (i.e., that have been recommended by ORNL for use in the EFPC assessment) fall within the 50th to 95th percentiles of the B_v distributions derived. In comparison, the NCRP values often exceeded the 100th percentile of the Belcher and Travis distributions.

For organic chemicals, point estimates for the BTFs were determined based on the octanol-water partition coefficient of each chemical and the relationships identified by Travis and Arms (1988). PDFs for select organic chemicals are available in the Belcher and Travis study and were used in this assessment.

5.2.3.4 Interpretation of PDFs for exposure variables

In reviewing the PDFs presented in Appendix O, it is apparent that the RME point estimates are generally quite conservative and fall in the upper tail of the probability distributions. This would be as expected given the desire of the regulatory agencies (in light of the inherent uncertainty in risk assessment) to err on the side of protection of human health. It is sufficient to note several key points:

- there is not one single "correct" value for each exposure variable used in risk characterization equations,
- every value selected for an exposure variable is actually a single point estimate within a distribution of possible values,
- the distribution of possible values for the exposure variable (i.e., the PDF) reflects the inherent uncertainty or variability in this factor,

- the PDF may be statistically evaluated to determine the percentile value associated with any point estimate in the distribution of possible values,
- the calculated percentile values associated with the RME point estimates provide an indication of the relative conservatism built into the point estimate,
- the uncertainty in each exposure variable, as characterized by the PDF, may be quantitatively combined to generate a true probabilistic estimate of risk to human health, and
- Monte Carlo simulation is a useful (and preferred) technique for combining the uncertainty in each input variable and understanding the influence on the final risk estimate.

Section 5.5 presents the results of the Monte Carlo simulation for critical exposure pathways of concern. This is the Tier III evaluation of risks to human health. In this section, the influence of parameter uncertainty on the final risk estimates is made explicit.

5.3 TOXICITY ASSESSMENT

The objective of this section is to evaluate the inherent toxicity of the COCs, and to identify and select toxicological measures needed for the BRA at EFPC. The toxicity measures are then combined with the results of the exposure assessment to estimate risk to receptors in the risk characterization section (Sect. 5.4).

At EFPC, health risks from both hazardous and radiological wastes are being evaluated. Therefore, toxicity information is provided for both chemical compounds and radionuclides.

Chemical Toxicity. The toxicity measures used in this risk assessment for chemical compounds are:

- RfDs for oral exposure - acceptable intake values for subchronic and chronic exposure (noncarcinogenic effects),
- RfCs for inhalation exposure - acceptable intake values for subchronic and chronic exposure (noncarcinogenic effects),
- carcinogenic slope factors for oral exposure, and
- carcinogenic slope factors for the inhalation route.

For noncarcinogens, EPA derives RfDs based on estimates of the no-observable-adverse-effect level (NOAEL) or lowest-observable-adverse-effect level (LOAEL) in humans or test animals using the following equation:

$$RFD = \frac{NOAEL}{(UF \times MF)} \qquad (31)$$

where

NOAEL = no-observable-adverse-effect-level (mg/kg body weight/d),
UF = uncertainty factor (unitless),
MF = modifying factor (unitless).

The NOAEL is the highest experimental dose at which there was no statistically significant increase in a toxicologically significant end point. Uncertainty factors (UFs) are intended to account for:

- variation in sensitivity among the members of the human population,
- uncertainty in extrapolating animal data to humans,
- uncertainty in extrapolation from data obtained in a study that is of less than lifetime exposure, and
- uncertainty in using LOAEL data rather than NOAEL data.

Commonly, each of these factors is set equal to 10. The modifying factor (MF) is an additional optionally used factor, the magnitude of which reflects professional judgment regarding the quality of the data used in the toxicological assessment (e.g., the completeness of the overall data base and the number of animals tested).

The inhalation RfC methodology requires conversion of the NOAEL levels observed in animals to human equivalent concentrations (HECs) before the data sets and effects levels can be evaluated and compared. The inhalation RfC is derived as follows:

$$RfC = \frac{NOAEL_{[HEC]}}{(UF \times MF)} \qquad (32)$$

where

$NOAEL_{[HEC]}$ = no-observable-adverse-effect-level (mg/kg body weight/d) adjusted to human equivalent concentration,
UF = uncertainty factor (unitless),
MF = modifying factor (unitless).

The $NOAEL_{[HEC]}$ is the key datum obtained from the evaluation of the dose-response relationship.

The inhalation RfCs are derived by EPA according to the Interim Methods for Development of Inhalation Reference Concentrations (EPA 1990e). These methods were developed by Agency scientists in the Office of Research and Development and peer-reviewed at a workshop/public meeting held at the EPA Environmental Research Center in Research Triangle Park, North Carolina on October 6, 1987. It was intended that these methods would be interim and that improvements in the supporting scientific data base and advancements in risk assessment extrapolation procedures would be incorporated on a regular basis. EPA is currently attempting to standardize its approach to determining RfCs. Final guidance has not yet been released by the Agency.

The assessment of the potential for noncarcinogenic effects (i.e., the use of RfDs and RfCs in risk assessment) is based on the assumption of a threshold below which adverse health effects are not anticipated to occur. Carcinogenesis, however, is generally thought to be a phenomenon for which the presumption of threshold effects is inappropriate (EPA 1989a). Therefore, EPA does not estimate an effects threshold for this class of chemicals. Alternately, EPA uses a two-part evaluation in which the subject chemical is first assigned a weight-of-evidence classification, and then a cancer potency (slope factor) is calculated.

The weight-of-evidence classification evaluates the evidence that a given chemical is a carcinogen in human and animal systems. These ratings are as follows:

- A: human carcinogen,
- B1: probable human carcinogen - limited human data are available,
  B2: probable human carcinogen - sufficient data in animals, and inadequate or no evidence in humans,
- C: possible human carcinogen,
- D: not classifiable as to human carcinogenicity, and
- E: evidence of noncarcinogenicity for humans.

EPA develops CSFs for carcinogens that have been rated A, B1, B2, and for many that have been rated C. The CSF is a plausible upper-bound estimate of the slope of the dose-response curve in the low dose range. It is interpreted as the probability of a cancer response per unit *intake* of a chemical over a lifetime. In risk assessment, the cancer potency factor is used to estimate the excess lifetime probability of a carcinogenic effect occurring in exposed receptors. Toxicity measures for carcinogens also can be expressed in terms of risk per unit concentration.

EPA has not developed slope factors or RfDs for the dermal exposure route. However, in the absence of these factors, EPA recommends that assessors use the oral toxicity values when calculating risks associated with the dermal route (EPA 1992b). This approach has been adopted in the current BRA. However, considerable uncertainty is associated with the use of oral measures for the dermal exposure pathway.

Most toxicity measures and intake estimates are expressed in terms of administered or applied dose [the amount of chemical at an exchange boundary (e.g., skin, lungs, gut) that is available for absorption]. There are two major exceptions. First, some toxicity measures *are* based on absorbed dose, not administered dose. However, this distinction is not usually specified, and the process of making Agency-reviewed adjustments (to convert to administered dose) is beyond the scope of this risk assessment. Therefore, no adjustments were made to compensate for toxicity values based on absorbed dose. Second, the dermal contact route, due to the incorporation of a relative absorption factor (for soils) or a permeability constant (for aqueous solutions), yields estimates of absorbed dose. Theoretically, toxicity values based on administered dose would then need to be converted to absorbed dose to calculate risk. However, the adjustment of the oral toxicity measure can be accomplished only if sufficient data are available in the principal laboratory studies on oral absorption efficiency in the species on which the toxicity measures are based. However, lacking the proper information to make the adjustment, EPA recommends using the unadjusted oral toxicity value (EPA 1992b). Therefore, no adjustments were made.

In January 1991, the decision was made to express inhalation toxicity values in terms of concentration in air (i.e., as RfCs and inhalation unit risks). This decision was based on two factors (EPA 1992e):

> ...1) the workgroups felt that it was technically more accurate to base toxicity values directly on measured air concentrations instead of making the metabolic, pharmacokinetic and /or surface area adjustments required to estimate an "internal dose"; and 2) there are compounds that elicit route-of-entry effects (e.g., sensitizers and irritants) where the toxic effect is to the respiratory system or exchange boundary where a measure of "internal dose" might inappropriately imply effects to other organ systems or effects from other exposure routes.

Therefore, in order to quantify risk according to presently accepted methodology (EPA 1989a), a conversion from air concentration to dose is necessary. For this reason, the Health Effects Assessment Summary Tables (HEAST) document contains inhalation slope factors for carcinogens [in units of $(mg/[kg \cdot d])^{-1}$] in which this conversion to dose has been made by the Carcinogen Risk Assessment Verification Endeavor (CRAVE) Work Group. For noncarcinogens, the conversion was made by multiplying by 20 m^3/day and dividing by 70 kg.

The valence state of chromium detections was not distinguished. Therefore, it has been assumed that all chromium is most likely present as trivalent chromium (Cr^{+3}).

EPA recommends the use of two primary sources of information for identifying and selecting toxicity measures: the IRIS data base (EPA 1993) and HEAST (EPA 1992e). Priority

is given to information contained within the IRIS data base. However, where IRIS does not contain needed information or toxicity measures, HEAST was consulted.

Radiation Effects. This risk assessment includes the potential for health effects from low-level exposure to ionizing radionuclide contaminants at EFPC. Primary health effects include an increase in the occurrence of cancer in specific organs, and possible genetic effects that may extend to future generations. Note, however, that noncarcinogenic effects (i.e., mutagenic and teratogenic) of radionuclide exposures are not considered in this assessment, and in all cases the cancer risks are assumed to prevail.

Radiation exposures for EFPC can be separated into two types: external or internal. External exposure occurs when the radioactive material is outside the body. Internal exposure occurs when the radioactive material enters the body by inhalation or ingestion. Inhaled material can be exhaled or expelled from the lungs, or deposited and absorbed into blood and relocated to other organs where it is excreted over time. Ingested material may enter the blood and be either excreted in the urine or feces or relocated to other organs and excreted over time. Most insoluble ingested material is not absorbed into the blood, but is excreted directly in the feces.

During the radioactive decay processes, alpha, beta, and gamma radiation may be released. Each type of radiation differs in its physical properties and in its ability to induce damage to biological tissue. Within the body, alpha particles cause the most damage because their energy is completely absorbed by surrounding tissue. Alpha particles are less of a hazard outside the body because they typically lose all of their energy in the dead skin cell layer of the body before reaching living tissue. Beta particles are also primarily an internal hazard; however, in cases of external skin exposure, very energetic beta particles can penetrate to living skin cells, thus representing an external hazard as well. Beta particles deposit less energy to small volumes of tissue and therefore induce much less damage than alpha particles. Gamma radiation is primarily an external hazard because it can penetrate tissue and reach internal organs. Gamma rays, however, tend to pass through and leave the body without depositing a large fraction of their energy.

Radiation health effects for humans have been confirmed at high doses for large populations. For low doses, health effects are statistically estimated from the high dose studies. Radiologic cancer risk estimates may be derived for either populations or individuals, but it is important to note that cancer risks (both nonradiologic and radiologic) are probabilistic estimates and thus can only theoretically apply to a given individual. Cancer risks are estimates of cancer incidence related to low-level doses, and are uncertain because of the underlying extrapolation from high doses. Studies of populations chronically exposed to low-level radiation, such as those residing in regions of elevated natural background, are not conclusive in providing evidence of an

associated increase in the risk of cancer. The science of carcinogenesis is continually evolving, and additional uncertainty is inherent because of the assumptions made concerning dose-response relationships and mechanisms of carcinogenesis.

Two methods for evaluating exposure to radionuclides have been identified by EPA (1989a). The first method, established by the International Commission on Radiological Protection (ICRP), estimates dose equivalents to specified organs and effective dose equivalent (for the whole body) due to intakes of radionuclides. Risk is then determined by multiplying the dose by the cancer risk incidence factor. In the second method, which is used in the EFPC risk assessment, intake estimates (in units of radionuclide activity) are multiplied by a route-specific CSF to yield risk. EPA has classified all radionuclides as Group A carcinogens based upon epidemiological studies of cancers in humans and animals.

EPA's Office of Radiation Programs has published ingestion, inhalation, and external exposure CSFs based upon compound-specific (e.g., radioactive, chemical, and metabolic) properties and information contained in reports from national and international scientific organizations [e.g., the National Academy of Sciences (NAS), the National Council on Radiation Protection and Measurement (NCRP), and ICRP]. The CSFs for the ingestion and inhalation routes (which are used in the BRA) are median or 50th percentile estimates of the age-averaged lifetime probability of a carcinogenic effect occurring in an exposed individual. These CSFs are taken from HEAST (EPA 1992e).

In the HEAST tables, certain radionuclides and radioactive decay chain products are designated with the suffix "+D," indicating that these radionuclides include the contributions from their decay products. Following guidance from ORNL, where available, slope factors with the "+D" suffix have been used in the EFPC BRA. They take into account decay of the radionuclide into its daughter products, the amount entered into the bloodstream via ingestion or inhalation, how the radionuclide is distributed to various body tissues and organs, and what dose is delivered. In addition, the age, weight, sex, and organ-specific factors of the receptor have been taken into account and do not need to be included in the estimate of exposure intake (EPA 1992e).

5.3.1 Toxicity Information for Noncarcinogenic and Carcinogenic Effects

This section contains toxicity information for the COCs at EFPC (previously identified in Sect. 5.1). In each profile, emphasis is placed on identifying the target effect of concern, the EPA basis for derivation of RfDs/RfCs and CSFs, and the weight-of-evidence and uncertainty associated with the EPA toxicity measures.

Each profile is divided into two sections: toxicity profile and uncertainty in toxicity measures. The first section contains the toxicity profile prepared by the Biomedical and Environmental Information Analysis (BEIA) section of the Health and Safety Research Division at ORNL (where available). These profiles were distributed to DOE contractors (August 10, 1992) to be used in all DOE Environmental Restoration Program risk assessments. **The information in this section has been taken verbatim from the ORNL compilation** except in cases where it was necessary to update the toxicity values based upon changes in IRIS and/or HEAST. For simplicity, when using the BEIA profiles, only the executive summary has been provided in the text below, and the complete, more detailed profile is contained in Appendix N. **In addition, due to the large number of references associated with each profile, references within each profile are included in Appendix N and not with the main text references.**

Note that BEIA profiles are not available for all COCs at EFPC. Therefore, BEIA profiles were used where available (inorganics), and profiles were developed for all other compounds (organics and radionuclides).

The second section (Uncertainty in Toxicity Measures) focuses on identifying uncertainty associated with the specific toxicity values and the studies upon which the values were derived. Information in this section is not a part of the BEIA profiles.

5.3.1.1 Antimony

Toxicity Profile

PHYSICOCHEMICAL CHARACTERISTICS							
A. Wt.	MP (°C)	BP (°C)	SG (@25°C)	Water Sol (mg/ℓ)	Log K_{oc}	Log K_{ow}	Vapor Pressure
121.75	630	1635	6,684	Insoluble	NA	NA	NA

NONCARCINOGENIC TOXICITY VALUES							
Oral RfD (mg/kg/d)		Uncertainty Factor		Inhal. RfC (mg/m³)		Uncertainty Factor	
Subchron	Chronic	Subchron	Chronic	Subchron	Chronic	Subchron	Chronic
4.00E-04	4.00E-04	1000	1000	NA	NA	NA	NA

CARCINOGENIC TOXICITY VALUES					
Cancer Slope Factor (CSF) (mg/kg/day)$^{-1}$		Unit Risk Factor (URF)		Weight-of-Evidence	Reference of Study (RfD/RfC/ CSF/URF)
Oral	Inhalation	Oral ($\mu g/\ell$)$^{-1}$	Inhalation ($\mu g/m^3$)$^{-1}$		
NA	NA	NA	NA	NA	Schroeder et al. 1970/NA/NA/ NA

Antimony (Sb) is a naturally occurring metal that is used in various manufacturing processes. It exists in valence states of 3 and 5 (Budavari 1989; ATSDR 1990). Antimony is a common urban air pollutant (Beliles 1979). Exposure to antimony may be via inhalation, oral and dermal routes (ATSDR 1990).

Antimony is sparingly absorbed following ingestion or inhalation (Felicetti et al. 1974a; Gerber et al. 1982; ATSDR 1990). Both gastrointestinal and pulmonary absorption are a function of compound solubility. Antimony is transported in the blood, its distribution varying among species and dependent on its valence state (Felicetti et al. 1974b). Antimony is not metabolized but may bind to macromolecules and react covalently with sulfhydryl and phosphate groups (ATSDR 1990). Excretion of antimony is primarily via the urine and feces, and also is dependent upon valence state (Cooper et al. 1968; Ludersdorf et al. 1987; ATSDR 1990).

Acute oral exposure of humans and animals to high doses of antimony or antimony-containing compounds (antimonials) may cause gastrointestinal disorders (vomiting, diarrhea), respiratory difficulties, and death at extremely high doses (Bradley and Frederick 1941; Beliles 1979; ATSDR 1990). Subchronic and chronic oral exposure may affect hematologic parameters (ATSDR 1990). Long-term exposure to high doses of antimony or antimonials has been shown to adversely affect longevity in animals (Schroeder et al. 1970). Limited data suggest that prenatal and postnatal exposure of rats to antimony interferes with vasomotor responses (Marmo et al. 1987; Rossi et al. 1987).

Acute inhalation exposure of humans may cause gastrointestinal disorders (probably due to ingestion of airborne antimony) (ATSDR 1990). Exposure of animals to high concentrations of antimony and antimonials (especially stibine gas) may result in pulmonary edema and death (Price et al. 1979). Long-term occupational exposure of humans has resulted in electrocardiac disorders, respiratory disorders, and possibly increased mortality (Renes 1953; Breiger et al. 1954). Antimony levels for these occupational exposure evaluations ranged from 2.2 to 11.98 mg Sb/m^3. Based on limited data, occupational exposure of women to metallic antimony and several antimonials has reportedly caused alterations in the menstrual cycle and an increased incidence of spontaneous abortions (Belyaeva 1967). Reproductive dysfunction has been demonstrated in rats exposed to antimony trioxide (Belyaeva 1967).

No data were available indicating that dermal exposure of humans to antimony or its compounds results in adverse effects. However dermal application of high doses of antimony oxide (1,584 mg Sb/kg) resulted in the death of rabbits within one day (IBTL 1972). Eye irritation due to exposure to stibine gas and several antimony oxides has been reported for humans (Stevenson 1965; Potkonjak and Pavlovich 1983).

The EPA (EPA 1991b) has calculated subchronic and chronic oral reference doses (RfDs) of 4E-4 mg/kg/day based on decreased longevity and alteration of blood chemistry in rats chronically exposed to potassium antimony tartrate in the drinking water (5 ppm equivalent to 0.35 mg Sb/kg/day). An uncertainty factor of 1,000 was applied: 10 for extrapolation from a lowest-observed-adverse-effect-level (LOAEL) to a no-observed-adverse-effect-level (NOAEL), 10 for extrapolation from animal data, and 10 for protection of sensitive populations.

The primary target organ for acute oral exposure to antimony appears to be the gastrointestinal tract (irritation, diarrhea, vomiting) and targets for long-term exposure are the blood (hematological disorders) and liver (mild hepatotoxicity) (ATSDR 1990). Inhalation exposure to antimony affects the respiratory tract (pneumoconiosis, restrictive airway disorders), with secondary targets being the cardiovascular system (altered blood pressure and electrocardiograms) and kidneys (histological changes) (Renes 1953; Breiger et al. 1954). Only limited evidence exists for reproductive disorders due to antimony exposure (Belyaeva 1967).

Although some data indicate that long-term exposure of rats to antimony trioxide and trisulfide increased the incidence of lung tumors (Wong et al. 1979; Watt 1980; Groth et al. 1986; Bio/dynamics 1989), the EPA has not evaluated antimony or antimonials for carcinogenicity and a Weight-of-Evidence classification is currently unavailable.

Uncertainty in Toxicity Measures

Noncarcinogenic. Uncertainty and Modifying Factors for the oral RfD are 1000 and 1, respectively. An uncertainty factor of 1000 (10 for inter-species conversion, 10 to protect sensitive individuals, and 10 because the effect level was a LOAEL and no NOEL was established) was applied to the LOAEL of 0.35 mg/kg. Confidence in the oral RfD is considered low because only one species was used, only one dose level was tested, no NOEL was determined, and gross pathology and histopathology were not well described. Confidence in the database is low due to lack of adequate oral exposure investigations. Low confidence in the RfD follows (USEPA 1985).

Carcinogenic. Quantitative estimates of carcinogenic risk for antimony by oral and inhalation exposure are not available from the EPA.

5.3.1.2 Arsenic

Toxicity Profile

PHYSICOCHEMICAL CHARACTERISTICS							
A. Wt.	MP (°C)	BP (°C)	SG (@25°C)	Water Sol (mg/L)	Log K_{oc}	Log K_{ow}	Vapor Pressure
74.92	817	613	5.73	Insoluble	NA	NA	0.00

NONCARCINOGENIC TOXICITY VALUES							
Oral RfD (mg/kg/d)		Uncertainty Factor		Inhal. RfC (mg/m^3)		Uncertainty Factor	
Subchron	Chronic	Subchron	Chronic	Subchron	Chronic	Subchron	Chronic
3.00E-04	3.00E-04	3	3	NA	NA	NA	NA

CARCINOGENIC TOXICITY VALUES					
Cancer Slope Factor (CSF) $(mg/kg/day)^{-1}$		Unit Risk Factor (URF)		Weight-of-Evidence	Reference of study (RfD/RfC/ CSF/URF)
Oral	Inhalation	Oral $(\mu g/L)^{-1}$	Inhalation $(\mu g/m^3)^{-1}$		
*1.75E+00	5.00E+01	5.00E-05	4.30E-03	A	Tseng 1977; NA;NA; Brown and Chu 1983 a,b,c

*This oral slope factor was calculated from the oral unit risk by assuming an ingestion of 2 liters of water per day by a 70-kg adult.

The toxicity of inorganic arsenic (As) depends on its valence state (-3, +3, or +5), and also on the physical and chemical properties of the compound in which it occurs. Trivalent (As^{+3}) compounds are generally more toxic than pentavalent (As^{+5}) compounds, and the more water soluble compounds are usually more toxic and more likely to have systemic effects than the less soluble compounds, which are more likely to cause chronic pulmonary effects if inhaled. One of the most toxic inorganic arsenic compounds is arsine gas (AsH_3). It should be noted that laboratory animals are generally less sensitive than humans to the toxic effects of inorganic arsenic. In addition, in rodents the critical effects appear to be immunosuppression and hepatorenal dysfunction, whereas in humans the skin, vascular system, and peripheral nervous system are the primary target organs.

Water soluble inorganic arsenic compounds are absorbed through the G.I. tract (>90%) and lungs; distributed primarily to the liver, kidney, lung, spleen, aorta, and skin; and excreted mainly in the urine at rates as high as 80% in 61 hr following oral dosing (U.S. EPA 1984; ATSDR 1989; Crecelius 1977). Pentavalent arsenic is reduced to the trivalent form and then methylated in the liver to less toxic methylarsenic acids (ATSDR 1989).

Symptoms of acute inorganic arsenic poisoning in humans are nausea, anorexia, vomiting, epigastric and abdominal pain, and diarrhea. Dermatitis (exfoliative erythroderma), muscle cramps, cardiac abnormalities, hepatotoxicity, bone marrow suppression and hematologic abnormalities (anemia), vascular lesions, and peripheral neuropathy (motor dysfunction, paresthesia) have also been reported (U.S. Air Force 1990; ATSDR 1989; Franzblau and Lilis 1989; EPA 1984; Armstrong et al. 1984; Hayes 1982; Mizuta et al. 1956). Oral doses as low as 20-60 μg/kg/day have been reported to cause toxic effects in some individuals (ATSDR 1989). Severe exposures can result in acute encephalopathy, congestive heart failure, stupor, convulsions, paralysis, coma, and death. The acute lethal dose to humans has been estimated to be about 0.6 mg/kg/day (ATSDR 1989). General symptoms of chronic arsenic poisoning in humans are weakness, general debility and lassitude, loss of appetite and energy, loss of hair, hoarseness of voice, loss of weight, and mental disorders (Hindmarsh and McCurdy 1986). Primary target organs are the skin (hyperpigmentation and hyperkeratosis) (Terada et al. 1960; Tseng et al. 1968; Zaldivar 1974; Cebrian et al. 1983; Huang et al. 1985), nervous system (peripheral neuropathy) (Hindmarsh et al. 1977, 1986; Valentine et al. 1982; Heyman et al. 1956; Mizuta et al. 1956; Tay and Seah 1975), and vascular system (Tseng et al. 1968; Borgano and Greiber 1972; Salcedo et al. 1984; Wu et al. 1989; Hansen 1990). Anemia, leukopenia, hepatomegaly, and portal hypertension have also been reported (Terada et al. 1960; Viallet et al. 1972; Morris et al. 1974; Datta 1976). In addition, possible reproductive effects include a high male to female birth ratio (Lyster 1977).

In animals, acute oral exposures can cause gastrointestinal and neurological effects (Heywood and Sortwell 1979). Oral LD_{50} values range from about 10 to 300 mg/kg (ATSDR 1989; U.S. Air Force 1990). Low subchronic doses can result in immunosuppression, (Blakely et al. 1980) and hepatorenal effects (Mahaffey et al. 1981; Brown et al. 1976; Woods and Fowler 1977, 1978; Fowler and Woods 1979; Fowler et al. 1979). Chronic exposures have also resulted in mild hyperkeratosis and bile duct enlargement with hyperplasia, focal necrosis, and fibrosis (Baroni et al. 1963; Byron et al. 1967). Reduction in litter size, high male/female birth ratios, and fetotoxicity without significant fetal abnormalities occur following oral exposures (Schroeder and Mitchener 1971; Hood et al. 1977; Baxley et al. 1981); however, parenteral dosing has resulted in exencephaly, encephaloceles, skeletal defects, and urogenital system abnormalities (Ferm and Carpenter 1968; Hood and Bishop 1972; Beaudoin 1974; Burk and Beandoin 1977).

The Reference Dose for chronic oral exposures, 0.0003 mg/kg/d, is based on a NOAEL of 0.0008 mg/kg/d and a LOAEL of 0.014 mg/kg/d for hyperpigmentation, keratosis, and possible vascular complications in a human population consuming arsenic-contaminated drinking water (EPA 1991a). Because of uncertainties in the data, EPA (1991a) states that "strong scientific arguments can be made for various values within a factor of 2 or 3 of the currently recommended

RfD value." The subchronic Reference Dose is the same as the chronic RfD, 0.0003 mg/kg/d (EPA 1992).

Acute inhalation exposures to inorganic arsenic can damage mucous membranes, cause rhinitis, pharyngitis and laryngitis, and result in nasal septum perforation (EPA 1984). Chronic inhalation exposures, as occurring in the workplace, can lead to rhino-pharyno-laryngitis, tracheobronchitis, (Lundgren 1954); dermatitis, hyperpigmentation, and hyperkeratosis (Perry et al. 1948; Pinto and McGill 1955); leukopenia (Kyle and Pease 1965; Hine et al. 1977); peripheral nerve dysfunction as indicated by abnormal nerve conduction velocities (Feldman et al. 1979; Blom et al. 1985; Landau et al. 1977); and peripheral vascular disorders as indicated by Raynaud's syndrome and increased vasospastic reactivity in fingers exposed to low temperatures (Lagerkvist et al. 1986). Higher rates of cardiovascular disease have also been reported in some arsenic-exposed workers (Lee and Fraumeni 1969; Axelson et al. 1978; Wingren and Axelson 1985). Possible reproductive effects include a high frequency of spontaneous abortions and reduced birth weights (Nordstrõm et al. 1978a,b). Arsine gas (AsH_3), at concentrations as low as 3-10 ppm for several hours, can cause toxic effects. Hemolysis, hemoglobinuria, jaundice, hemolytic anemia, and necrosis of the renal tubules have been reported in exposed workers (ACGIH 1986; Fowler and Weissberg 1974).

Animal studies have shown that inorganic arsenic, by intratracheal instillation, can cause pulmonary inflammation and hyperplasia (Webb et al. 1986, 1987), lung lesions (Pershagen et al. 1982), and immunosuppression (Hatch et al. (1985). Long-term inhalation exposures have resulted in altered conditioned reflexes and CNS damage (Rozenshstein 1970). Reductions in fetal weight and in the number of live fetuses, and increases in fetal abnormalities due to retarded osteogenesis have been observed following inhalation exposures (Nagymajtenyi et al. 1985).

Subchronic and chronic RfCs for inorganic arsenic have not been derived.

Epidemiological studies have revealed an association between arsenic concentrations in drinking water and increased incidences of skin cancers (including squamous cell carcinomas and multiple basal cell carcinomas), as well as cancers of the liver, bladder, respiratory and gastrointestinal tracts (EPA 1987; IARC 1987; Sommers et al. 1953; Reymann et al. 1978; Dobson et al. 1965; Chen et al. 1985, 1986). Occupational exposure studies have shown a clear correlation between exposure to arsenic and lung cancer mortality (IARC 1987; EPA 1991a). U.S. EPA (1991a) has placed inorganic arsenic in weight-of-evidence group A, human carcinogen. A drinking water unit risk of 5E-5$(\mu g/L)^{-1}$ has been proposed (EPA 1991a); this was derived from drinking water unit risks for females and males that are equivalent to slope factors of 1.0E-3 $(\mu g/kg/d)^{-1}$ (females) and 2.0E-3 $(\mu g/kg/d)^{-1}$ (males) (EPA 1987). For inhalation

exposures, a unit risk of 4.3E-3 $(\mu g/m^3)^{-1}$ (EPA 1991a) and a slope factor of 5.0E+1 $(mg/kg/d)^{-1}$ have been derived (EPA 1992).

Uncertainty in Toxicity Measures

Noncarcinogenic. An uncertainty factor of 3 was used to derive the oral RfD to account for both the lack of data to preclude reproductive toxicity as a critical effect and to account for some uncertainty in whether the NOAEL of the critical study accounts for all sensitive individuals. The modifying factor for the oral RfD is one.

The confidence in the chosen study is considered medium (EPA 1991a). An extremely large number of people were included in the assessment (>40,000), but the doses were not well-characterized and other contaminants were present. The supporting human toxicity data base is extensive, but somewhat flawed, and problems exist with all of the epidemiological studies.

Carcinogenic. The inhalation unit risk for arsenic was estimated as 4.3×10^{-3} $(\mu g/m^3)^{-1}$ based on human lung cancer in males following occupational exposure via inhalation (Brown and Chu 1983a,b,c; Lee-Feldstein 1983). A geometric mean was obtained for data sets obtained within distinct exposed populations (EPA 1984a). The final estimate is the geometric mean of those two values. It was assumed that the increase in the age-specific mortality rate of lung cancer was a function only of cumulative exposure (EPA 1991a).

In the discussion of confidence for inhalation exposure resulting in carcinogenicity, the overall confidence was high (EPA 1991a). A large study population was observed. Exposure assessments included air measurements for the Anaconda smelter and both air measurements and urinary arsenic for the ASARCO smelter. Observed lung cancer incidence was significantly increased over the expected values. The range of the estimates derived from data from two different exposure areas was within a factor of 6.

5.3.1.3 Barium

Toxicity Profile

PHYSICOCHEMICAL CHARACTERISTICS							
A. Wt.	MP (°C)	BP (°C)	SG (@25°C)	Water Sol (mg/ℓ)	Log K_{oc}	Log K_{ow}	Vapor Pressure
137.33	710	1600	3.6	NA	NA	NA	10

NONCARCINOGENIC TOXICITY VALUES							
Oral RfD (mg/kg/d)		Uncertainty Factor		Inhal. RfC (mg/m³)		Uncertainty Factor	
Subchron	Chronic	Subchron	Chronic	Subchron	Chronic	Subchron	Chronic
7.00E-02	7.00E-02	3	3	5.00E-03	5.00E-04	100	1000

CARCINOGENIC TOXICITY VALUES					
Cancer Slope Factor (CSF) (mg/kg/day)$^{-1}$		Unit Risk Factor (URF)		Weight-of-Evi-dence	Reference of Study(RfD-/RfC/CSF/U-RF)
Oral	Inhalation	Oral ($\mu g/\ell$)$^{-1}$	Inhalation ($\mu g/m^3$)$^{-1}$		
NA	NA	NA	NA	NA	Wones et al. 1990/Brenniman and Levy 1984/Taraseko et al. 1970/NA

The soluble salts of barium, an alkaline earth metal, are toxic in mammalian systems. They are absorbed rapidly from the gastrointestinal tract and are deposited in the muscles, lungs and bone. Barium is excreted primarily in the feces.

At low doses, barium acts as a muscle stimulant and at higher doses affects the nervous system eventually leading to paralysis. Acute and subchronic oral doses of barium cause vomiting and diarrhea, followed by decreased heart rate and elevated blood pressure. Higher doses result in cardiac irregularities, weakness, tremors, anxiety and dyspnea. A drop in serum potassium may account for some of the symptoms. Death can occur from cardiac and respiratory failure. Acute doses around 0.8 grams can be fatal to humans.

Subchronic and chronic oral or inhalation exposure primarily affects the cardiovascular system resulting in elevated blood pressure. A LOAEL of 0.51 mg barium/kg/day based on increased blood pressure was observed in chronic oral rat studies (Perry et al. 1983), whereas human studies identified a NOAEL of 0.21 mg barium/kg/day (Wones et al. 1990; Brenniman and Levy 1984). The human data were used by the EPA to calculate a chronic and subchronic oral reference dose (RfD) of 0.07 mg/kg/day. In the Wones et al. study, human volunteers were

given barium up to 10 mg/L in drinking water for 10 weeks. No clinically significant effects were observed. An epidemiological study was conducted by Brenniman and Levy in which human populations ingesting 2 to 10 mg/L of barium in drinking water were compared to a population ingesting 0 to 0.2 mg/L. No significant individual differences were seen; however, a significantly higher mortality rate from all combined cardiovascular diseases was observed with the higher barium level in the 65+ age group. The average barium concentration was 7.3 mg/L, which corresponds to a dose of 0.20 mg/kg/day. Confidence in the oral RfD is rated medium by the EPA.

Subchronic and chronic inhalation exposure of human populations to barium-containing dust can result in a benign pneumoconiosis called "baritosis." This condition is often accompanied by an elevated blood pressure but does not result in a change in pulmonary function. Exposure to an air concentration of 5.2 mg/m^3 for 4 hours/day for 6 months has been reported to result in elevated blood pressure and decreased body weight gain in rats (Tarasenko et al., 1977). Reproduction and developmental effects were also observed. Increased fetal mortality was seen after untreated females were mated with males treated with 5.2 mg/m^3 of barium. Similar results were obtained with female rats treated with 13.4 mg/m^3. The NOAEL for developmental effects was 1.15 mg/m^3. An inhalation reference concentration (RfC) of 0.005 mg/m^3 for both subchronic and chronic exposure was calculated by the EPA based on the NOAEL for developmental effects. These effects have not been substantiated in humans or other animal systems.

The EPA weight-of-evidence classification for cancer is D, not classifiable as to human carcinogenicity based on inadequate evidence in humans and animals.

Uncertainty in Toxicity Measures

Noncarcinogenic. An uncertainty factor of 3 was applied when calculating the lifetime oral RfD (USEPA 1990). The EPA determined that because of both the critical study's unique focus and the supporting studies, a 3-fold uncertainty factor, instead of 10-fold, was chosen as most appropriate to protect for sensitive individuals within that population. EPA believes that medium confidence can be placed in the total data base used to determine the chronic oral RfD (USEPA 1990). A subchronic and chronic inhalation RfC is available in HEAST. The corresponding uncertainty factors are 100 and 1000, respectively.

Carcinogenic. Quantitative estimates of carcinogenic risk for barium by oral and inhalation exposure are not available from the EPA.

5.3.1.4 Beryllium

Toxicity Profile

PHYSICOCHEMICAL CHARACTERISTICS							
A. Wt.	MP (°C)	BP (°C)	SG (@25°C)	Water Sol (mg/L)	Log K_{oc}	Log K_{ow}	Vapor Pressure
9.01	1278	2970	1.85	Insoluble	NA	NA	0.00

NONCARCINOGENIC TOXICITY VALUES							
Oral RfD (mg/kg/d)		Uncertainty Factor		Inhal. RfC (mg/m^3)		Uncertainty Factor	
Subchron	Chronic	Subchron	Chronic	Subchron	Chronic	Subchron	Chronic
5.00E-03	5.00E-03	100	100	NA	NA	NA	NA

CARCINOGENIC TOXICITY VALUES					
Cancer Slope Factor (CSF) $(mg/kg/d)^{-1}$		Unit Risk Factor (URF)		Weight-of-Evidence	Reference of study (RfD/RfC/ CSF/URF)
Oral	Inhalation	Oral $(\mu g/L)^{-1}$	Inhalation $(\mu g/m^3)^{-1}$		
4.30E+00	8.40E+00	1.20E-04	2.40E-03	B2	Schroeder & Mitchner 1975a; NA; Schroeder & Mitchner 1975a; Wagoner et al. 1980

Beryllium is present in the earth's crust, in emissions from coal combustion, in surface water and soil, and in house dust, food, drinking water, and cigarette smoke (EPA 1987a). However, the highest risk for exposure occurs among workers employed in beryllium manufacturing, fabricating, or reclamation industries (ATSDR 1988). Workers encounter dusts and fumes of many different beryllium compounds; the current occupational standard for worker exposure to beryllium is 2 $\mu g/m^3$ during an 8-hour workshift (OSHA 1989).

Inhaled beryllium is absorbed slowly and localizes mainly in the lungs, bone, liver and kidneys (Stiefel et al. 1980; Reeves et al. 1967; Reeves and Vorwald 1967; Zorn et al. 1988; Tepper et al. 1961; Meehan and Smyth 1967). Ingested beryllium undergoes limited absorption and localizes in liver, kidneys, lungs, stomach, spleen and the large and small intestines (Crowley et al. 1949; Furchner et al. 1973; Watanabe et al. 1985). Significant absorption of beryllium or its compounds through intact skin is unlikely because of its chemical properties (EPA 1987b). Beryllium per se is not biotransformed, but soluble salts may be converted to less soluble compounds in the lung (EPA 1987b). Most orally administered beryllium passes through the

gastrointestinal tract unabsorbed and is excreted in the feces (Reeves 1965), whereas inhaled water-soluble beryllium salts are excreted mainly by the kidneys (Zorn et al. 1988).

Limited data indicate that the oral toxicity of beryllium is low. No adverse effects were noted in mice given 5 ppm beryllium in the drinking water in a lifetime bioassay (Schroeder and Mitchener 1975a,b). The dose (converted to 0.54 mg/kg bw/d) was the no-adverse-effect level (NOAEL) used in the calculation of the chronic oral RfD for beryllium of 0.005 mg/kg/d (EPA 1991a).

In contrast, the toxicity of inhaled beryllium is well-documented. Humans inhaling "massive" doses of beryllium compounds (such as the water soluble sulfate, fluoride, chloride, and oxide) may develop acute berylliosis (Constantinidis 1978). ATSDR (1988) estimated that, based on existing data, the disease could develop at levels ranging from approximately 2-1000 μg Be/m^3. This disease usually develops shortly after exposure and is characterized by rhinitis, pharyngitis, and/or tracheobronchitis, and may progress to severe pulmonary symptoms. The severity of acute beryllium toxicity correlates with exposure levels, and the disease is now rarely observed in the United States because of improved industrial hygiene (Zorn et al. 1988; Kriebel et al. 1988b).

Humans inhaling beryllium may also develop chronic berylliosis which, in contrast to acute berylliosis, is highly variable in onset, is more likely to be fatal, and can develop a few months to $\geq$20 years after exposure (Constantinidis 1978; Hall et al. 1959; Kriebel et al. 1988b). Chronic beryllium disease is a systemic disease that primarily affects the lungs and is characterized by the development of noncaseating granulomas. The disease most likely results from a hypersensitivity response to beryllium as evidenced by positive patch tests (Nishimura 1966) and positive lymphocyte transformation tests (Williams and Williams (1983) in exposed individuals. Granulomas may also appear in the skin, liver, spleen, lymph nodes, myocardium, skeletal muscles, kidney, bone, and salivary glands (Kriebel et al. 1988b; Freiman and Hardy 1970).

Epidemiologic studies have suggested that beryllium and its compounds could be human carcinogens. In a study that covered 15 regions of the U.S., Berg and Burbank (1972) found a significant correlation between cancers of the breast, bone and uterus and the concentration and detection frequency of beryllium in drinking water. However, imperfect analytical and sampling methods used in the study prompted the EPA (1986b) to conclude that these results are not proof of cause and effect relationships between cancer and beryllium in drinking water. Studies in workers exposed to beryllium, mostly via inhalation, have shown significant increases in observed over expected lung cancer incidences (Bayliss et al. 1971; Bayliss and Lainhart 1972; Bayliss and Wagoner 1977; Wagoner et al. 1980; Mancuso 1970; 1979; 1980). The EPA (1986a), in

evaluating the total database for the association of lung cancer with occupational exposure to beryllium, noted several limitations, but concluded that the results must be considered to be at least suggestive of a carcinogenic risk to humans. In laboratory studies, beryllium sulfate caused increased incidences of pulmonary tumors in rats and rhesus monkeys (Vorwald 1953, 1962, 1968; Vorwald et al. 1955, 1966; Schepers et al. 1957; Reeves and Deitch 1969).

Based on sufficient evidence for animals and inadequate evidence for humans, beryllium has been placed in the EPA weight-of-evidence classification B2, probable human carcinogen (EPA 1991a). For inhalation exposure, the unit risk value is 2.4E-3 $(\mu g/m^3)^{-1}$, and the slope factor is 8.4 $(mg/kg/d)^{-1}$ (EPA 1991b). For oral exposure, the unit risk value is 1.2E-4 $(\mu g/L)^{-1}$ and the slope factor is 4.3 $(mg/kg/d)^{-1}$ (EPA 1991a).

Uncertainty in Toxicity Measures

Noncarcinogenic. A NOAEL of 0.54 mg per kg bw per day was used to calculate the chronic oral RfD of 0.005 mg per kg per day from a lifetime drinking water study in rats (Schroeder and Mitchener 1975a). The uncertainty factor of 100 reflects a factor of 10 each for interspecies conversion and the protection of sensitive human subpopulations (EPA 1991a). The oral RfD modifying factor is one.

Confidence in the study is rated as low because only one dose level was administered. Although numerous inhalation investigations and a supporting chronic oral bioassay in mice exist, these studies are considered as low to medium quality; thus, the data base is given a low confidence rating. The overall confidence in the RfD is low, reflecting the need for more toxicity data by the oral route.

Carcinogenic. The quantitative estimate of carcinogenic risk from oral exposure (oral slope factor) is 4.3 mg per kg per day. Drinking water unit risk is estimated at 1.2×10^{-4} per $\mu g/L$ (Schroeder and Mitchener 1975a). However, it is important to note that the unit risk should not be used if the water concentration exceeds 83 $\mu g/L$, since the slope factor may differ from that stated above this concentration.

The level of confidence for oral exposure may be considered limited due to high mortality and unspecified type and site of the tumors. Further, the estimate is based on a study that did not show a significant increase in tumorigenic response and the study has only one nonzero dose group (EPA 1991a).

The quantitative estimate of carcinogenic risk from inhalation exposure (inhalation slope factor) is 8.4 per mg per kg per day (EPA 1991a). Inhalation unit risk is estimated at 2.4×10^{-3} per $\mu g/m^3$. Both of the risk estimates are based on the relative risk extrapolation method. Despite several limitations, human data were used to quantify inhalation exposure (Wagoner et al. 1980). Humans are most likely to be exposed by inhalation to beryllium oxide, rather than other beryllium salts. Animal studies by inhalation of beryllium oxide have utilized intratracheal instillation rather than general inhalation exposure.

The estimates of exposure levels and duration are somewhat uncertain in these studies. While a quantitative assessment based on several animal studies resulted in a similar estimate of risk (which probably increases the confidence), the quality of the available data is considered poor, since the data lacked multi-dosed studies and adequate controls (EPA 1991a).

5.3.1.5 Cadmium

Toxicity Profile

PHYSICOCHEMICAL CHARACTERISTICS							
A. Wt.	MP (°C)	BP (°C)	SG (@25°C)	Water Sol (mg/L)	Log K_{oc}	Log K_{ow}	Vapor Pressure
112.41	321	765	8.64	Insoluble	NA	NA	0.00

NONCARCINOGENIC TOXICITY VALUES							
Oral RfD (mg/kg/d)		Uncertainty Factor		Inhal. RfC (mg/m^3)		Uncertainty Factor	
Subchron	Chronic	Subchron	Chronic	Subchron	Chronic	Subchron	Chronic
NA	5.00E-04 (water) 1.00E-03 (food)	NA	10 10	NA	NA	NA	NA

CARCINOGENIC TOXICITY VALUES					
Cancer Slope Factor (CSF) $(mg/kg/d)^{-1}$		Unit Risk Factor (URF)		Weight-of-Evidence	Reference of study (RfD/RfC/ CSF/URF)
Oral	Inhalation	Oral $(\mu g/L)^{-1}$	Inhalation $(\mu g/m^3)^{-1}$		
NA	6.10E+00	NA	1.80E-03	B1	EPA 1985a; NA; EPA 1985; EPA 1985

Cadmium is a naturally occurring metal that is used in various chemical forms in metallurgical and other industrial processes, and in the production of pigments. Environmental exposure can occur via the diet and drinking water (ATSDR 1989).

Cadmium is absorbed more efficiently by the lungs (30 to 60%) than by the gastrointestinal tract, the latter being a saturable process (Nordberg et al. 1985). Cadmium is transported in the blood and widely distributed in the body but accumulates primarily in the liver and kidneys (Goyer 1991). Cadmium burden (especially in the kidneys and liver) tends to increase in a linear fashion up to about 50 or 60 years of age after which the body burden remains somewhat constant. Metabolic transformations of cadmium are limited to its binding to protein and nonprotein sulfhydryl groups, and various macromolecules, such as metallothionein, which is especially important in the kidneys and liver (ATSDR 1989). Cadmium is excreted primarily in the urine.

Acute oral exposure to 20-30 g have caused fatalities in humans. Exposure to lower amounts may cause gastrointestinal irritation, vomiting, abdominal pain, and diarrhea (ATSDR 1989). An asymptomatic period of one-half to one hour may precede the onset of clinical signs. Oral LD_{50} values in animals range from 63 to 1125 mg/kg, depending on the cadmium compound (USAF 1990). Longer term exposure to cadmium primarily affects the kidneys, resulting in tubular proteinosis although other conditions such as "itai-itai" disease may involve the skeletal system. Cadmium involvement in hypertension is not fully understood (Goyer 1991).

Inhalation exposure to cadmium and cadmium compounds may result in effects including headache, chest pains, muscular weakness, pulmonary edema, and death (USAF 1990). The 1-minute and 10-minute lethal concentration of cadmium for humans has been estimated to be about 2,500 and 250 mg/m^3, respectively (Barrett et al. 1947; Beton et al. 1966). An 8-hour TWA (time-weighted-average) exposure level of 5 mg/m^3 has been estimated for lethal effects of inhalation exposure to cadmium, and exposure to 1 mg/m^3 is considered to be immediately dangerous to human health (Friberg 1950). Renal toxicity (tubular proteinosis) may also result from inhalation exposure to cadmium (Goyer 1991).

Chronic oral RfDs of 5E-4 and 1E-3 mg/kg/day have been established for cadmium exposure via drinking water and food, respectively (EPA 1991). Both values reflect incorporation of an uncertainty factor of 10. The RfDs are based on an extensive data base regarding toxicokinetics and toxicity in both human and animals, the critical effect being renal tubular proteinuria. Confidence in the RfD and data base is high.

Inhalation RfC values are currently not available.

The target organ for cadmium toxicity via oral exposure is the kidney (Goyer 1991). For inhalation exposure, both the lungs and kidneys are target organs for cadmium-induced toxicity (ATSDR 1989; Goyer 1991).

There is limited evidence from epidemiologic studies for cadmium-related respiratory tract cancer (ATSDR 1989). An inhalation unit risk of 1.8E-3 $(\mu g/m^3)^{-1}$ and an inhalation slope factor of 6.1E+0 $(mg/kg/d)^{-1}$ are based on respiratory tract cancer associated with occupational exposure (EPA 1985). Based on limited evidence from multiple occupational exposure studies and adequate animal data, cadmium is placed in weight-of-evidence group B1--probable human carcinogen.

Uncertainty in Toxicity Measures

Noncarcinogenic. The RfDs for chronic oral exposure for cadmium are 0.0005 mg per kg per day (NOAEL, water) and 0.001 mg per kg per day (NOAEL, food) (EPA 1993). EPA determined that a concentration of 200 μg cadmium (Cd/gm wet human renal cortex) is the highest renal level not associated with significant proteinuria. Using a toxicokinetic model, which assumes 0.01% elimination of cadmium body burden per day, and 2.5% absorption of cadmium from food or 5% from water, the predicted NOAEL for chronic cadmium exposure is 0.005 and 0.01 mg per kg per day from water and food, respectively.

The uncertainty factor for the oral RfD was estimated at 10. This is based on intrahuman variability to cadmium toxicity due to a lack of specific data on sensitive populations (EPA 1985a). The oral RfD modifying factor is one.

When considering the confidence in the oral RfD, it is important to note that a toxicokinetic model has been used to determine the highest level of exposure associated with the lack of a critical effect. The choice of a NOAEL does not reflect the information from any single study, but reflects the data obtained from many studies on the toxicity of cadmium in both humans and animals. These data also permit calculation of pharmacokinetic parameters of cadmium absorption, distribution, metabolism, and elimination. All of this information considered together gives high confidence in the data base. High confidence in either RfD follows as well.

Carcinogenic. Data are currently insufficient to classify cadmium as carcinogenic to humans by the oral route. The carcinogenic inhalation slope factor of 6.1 per mg per kg per day and the inhalation unit risk of 1.8×10^{-3} per $\mu g/m^3$ (EPA 1985a) were obtained by a two-stage method where only the first stage is affected by exposure.

The confidence in the carcinogenicity data is rated as moderate. The data were derived from a relatively large cohort. Effects of arsenic and smoking were accounted for in the quantitative analysis for cadmium effects. A slope factor derived from cadmium chloride inhalation assay data in rats (Takenaka et al. 1983) equals 3.4×10^{-1} per μg per kg per day for elemental cadmium or 2.1×10^{-1} per μg per kg per day for cadmium chloride. However, it was felt that the use of available human data was more reliable because of species variations in response and the type of exposure (cadmium salt versus cadmium fume and cadmium oxide).

5.3.1.6 Chromium

Toxicity Profile

PHYSICOCHEMICAL CHARACTERISTICS							
A. Wt.	MP (°C)	BP (°C)	SG (@25°C)	Water Sol (mg/L)	Log K_{oc}	Log K_{ow}	Vapor Pressure
52.00	1900	2642	7.14	Insoluble	NA	NA	NA

NONCARCINOGENIC TOXICITY VALUES							
Oral RfD (mg/kg/d)		Uncertainty Factor		Inhal. RfC (mg/m³)		Uncertainty Factor	
Subchron	Chronic	Subchron	Chronic	Subchron	Chronic	Subchron	Chronic
1.00E+00 (CrIII) 2.00E-02 (CrVI)	1.00E+00 (CrIII) 5.00E-03 (CrVI)	1000 (CrIII) 100 (CrVI)	100 (CrIII) 500 (CrVI)	NA NA	NA NA	NA NA	NA NA

CARCINOGENIC TOXICITY VALUES					
Cancer Slope Factor (CSF) $(mg/kg/d)^{-1}$		Unit Risk Factor (URF)		Weight-of-Evidence	Reference of study (RfD/RfC/ CSF/URF)
Oral	Inhalation	Oral $(\mu g/L)^{-1}$	Inhalation $(\mu g/m^3)^{-1}$		
NA NA	NA (CrIII) 4.10E+01 (CrVI)	NA NA	NA (CrIII) 1.2E-02 (CrVI)	NA (CrIII) A (CrVI)	EPA 1991a,b,c; NA; EPA 1991a; EPA 1991a

Elemental chromium (Cr) does not occur in nature, but is present in ores, primarily chromite ($FeOCr_2O_3$) (Hamilton and Wetterhahn 1988). Only two of the several oxidation states of chromium, Cr(III) and Cr(VI), are reviewed in this report based on their predominance and stability in the ambient environment and their toxicity in humans and animals.

Chromium plays a role in glucose and cholesterol metabolism and is thus an essential element to man and animals (Schroeder et al. 1962). Nonoccupational exposure to the metal

occurs via the ingestion of chromium-containing food and water, whereas occupational exposure occurs via inhalation (Langard 1982; Pedersen 1982). Workers in the chromate industry have been exposed to estimated chromium levels of 10-50 $\mu g/m^3$ for Cr(III) and 5-1000 $\mu g/m^3$ for Cr(VI); however, improvements in the newer chrome-plating plants have reduced the Cr(VI) concentrations 10- to 40-fold (Stern 1982).

Chromium(III) is poorly absorbed, regardless of the route of exposure, whereas chromium(VI) is more readily absorbed (Hamilton and Wetterhahn 1988). Humans and animals localize chromium in the lung, liver, kidney, spleen, adrenals, plasma, bone marrow, and red blood cells (RBC) (Langard 1982; ATSDR 1989; Bragt and van Dura 1983; Hamilton and Wetterhahn 1988). There is no evidence that chromium is biotransformed, but Cr(VI) does undergo enzymatic reduction, resulting in the formation of reactive intermediates and Cr(III) (Hamilton and Wetterhahn 1988). The main routes for the excretion of chromium are via the kidneys/urine and the bile/feces (Guthrie 1982; Langard 1982).

Animal studies show that Cr(VI) is generally more toxic than Cr(III), but neither oxidation state is very toxic by the oral route. In long-term studies, rats were not adversely affected by $\sim$1.9 g/kg/d of chromic oxide [Cr(III)] (diet), 2.4 mg/kg/d of Cr(III) as chromic chloride (drinking water), or 2.4 mg/kg/d of Cr(VI) as potassium dichromate (drinking water) (Ivankovic and Preussmann 1975; MacKenzie et al. 1958).

The respiratory and dermal toxicity of chromium are well-documented. Workers exposed to chromium have developed nasal irritation (at <0.01 mg/m^3, acute exposure), nasal ulcers, perforation of the nasal septum (at ~ 2 $\mu g/m^3$, subchronic or chronic exposure) (Hamilton and Wetterhahn 1988; ATSDR 1989; Lindberg and Hedenstierna 1983) and hypersensitivity reactions and "chrome holes" of the skin (Pedersen 1982; Burrows 1983; U.S Air Force 1990). Among the general population, contact dermatitis has been associated with the use of bleaches and detergents (Love 1983).

Compounds of both Cr(VI) and Cr(III) have induced developmental effects in experimental animals that include neural tube defects, malformations, and fetal deaths (Iijima et al. 1983; Danielsson et al. 1982; Matsumoto et al. 1976).

Subchronic and chronic oral RfD values are 1 mg/kg/d for Cr(III) and 0.02 and 0.005 mg/kg/d, respectively, for Cr(VI) (EPA 1991a,b,c). The subchronic and chronic oral RfD values for Cr(VI) and Cr(III) are derived from no-observed-adverse-effect levels (NOAELs) of 1.47 g/kg Cr(III)/day and 25 ppm of potassium dichromate [Cr(VI)] in drinking water, respectively

(Ivankovic and Preussmann 1975; MacKenzie et al. 1958). The inhalation RfC values for both Cr(III) and Cr(VI) are currently under review by an EPA workgroup.

The inhalation of chromium compounds has been associated with the development of cancer in workers in the chromate industry. The relative risk for developing lung cancer has been calculated to be as much as 30 times that of controls (Hayes 1982; Leonard and Lauwerys 1980; Langard 1983). There is also evidence for an increased risk of developing nasal, pharyngeal, and gastrointestinal carcinomas (Hamilton and Wetterhahn 1988). Quantitative epidemiological data were obtained by Mancuso and Hueper (1951), who observed an increase in deaths (18.2%; $p < 0.01$) from respiratory cancer among chromate workers compared with 1.2% deaths among controls. In a follow-up study, conducted when more than 50% of the cohort had died, the observed incidence for lung cancer deaths had increased to approximately 60% (Mancuso, 1975). The workers were exposed to 1-8 mg/m^3/year total chromium. Mancuso (1975) observed a dose response for total chromium exposure and attributed the lung cancer deaths to exposure to insoluble [Cr(III)], soluble [Cr(VI)], and total chromium. The results of inhalation studies in animals have been equivocal or negative (Nettesheim et al. 1971; Glaser et al 1986; Baetjer et al. 1959; Steffee and Baetjer 1965).

Based on sufficient evidence for humans and animals, Cr(VI) has been placed in the EPA weight-of-evidence classification A, human carcinogen (EPA 1991a). For inhalation exposure, the unit risk value is 1.2E-2 $(\mu g/m^3)^{-1}$ and the slope factor is 4.1E+01 $(mg/kg/d)^{-1}$ (EPA 1991a).

Uncertainty in Toxicity Measures

Noncarcinogenic. The biological action of chromium is dependent upon its valence. However, since hexavalent chromium is reduced to the trivalent state upon contact with biological material during its passage through membranes and cells, and also to some extent in the gastrointestinal tract, it may be difficult to toxicologically distinguish between the effects of these two oxidation states (EPA 1984a). The chronic oral RfD for hexavalent chromium of 0.005 mg per kg per day was derived from a 1-year drinking study of rats (MacKenzie et al. 1958). No effects were reported at 25 mg/L of chromium as K_2CrO_4 for 1 year (converted to 2.4 mg of chromium (VI) per kg per day assuming drinking water consumption was 0.097 L per kg per day). The chronic oral RfD for trivalent chromium is one. This value was derived from a chronic feeding study of rats in which a 5% mixture of Cr_2O_3 was included in the diet for 5 days per week for 600 feedings. In HEAST, subchronic RfDs are listed as 2.0×10^{-2} and 1 for hexavalent and trivalent chromium, respectively (EPA 1991b).

The chronic oral RfD for hexavalent chromium is limited to metallic chromium (IV) of soluble salts. Examples of soluble salts include potassium dichromate ($K_2Cr_2O_7$), sodium dichromate ($Na_2Cr_2O_7$), potassium chromate (K_2CrO_4), and sodium chromate (Na_2CrO_4) (EPA 1991a). The chronic oral RfD for trivalent chromium is limited to metallic chromium (III) of insoluble salts. Examples of insoluble salts include chromic (III) oxide (Cr_2O_3) and chromium (III) sulfate [$Cr_2(SO_4)_3$].

The uncertainty factors of 500 and 100 used in deriving the chronic RfDs for chromium (VI) and chromium (III), respectively, represent two 10-fold decreases in dose to account for both the expected interhuman and interspecies variability in the toxicity of the chemical in lieu of specific data. For chromium (VI), an additional factor of 5 was added to compensate for the less-than-lifetime exposure duration of the principal study (EPA 1991a,c). The uncertainty factors from HEAST for the subchronic toxicity values are 100 and 1,000 for chromium (VI) and chromium (III), respectively.

The oral RfD modifying factor for hexavalent chromium is one. The modifying factor of 10 for trivalent chromium reflects uncertainty in the NOAEL because the effects observed in the 90-day study were not explicitly addressed in the 2-year study, and thus, the highest NOAEL in the 2-year study may be a LOAEL; the absorption of chromium is low (< 1%) and is influenced by a number of factors, and thus, a considerable potential variation in absorption exists; and animals were allowed to die naturally after feeding stopped (2 years) and only then was histology performed (EPA 1991a,c).

Confidence in the study for chromium (VI) is low due to the small number of animals tested and parameters measured, and the lack of toxic effect at the highest dose tested. Confidence in the data base is low as the supporting studies are of low quality, and teratogenic and reproductive endpoints are not well-studied. For chromium (III), the principal study is rated low due to the lack of explicit detail on study protocol and results. The confidence in the data base is low due to the lack of high-dose supporting data. The low confidence in the RfD also reflects the lack of an observed effect level (EPA 1991a,c).

Carcinogenic. Chromium (VI) is classified by EPA under category A, meaning it is a human carcinogen (EPA 1991a). Dose-response relationships have been established for chromium-induced lung cancer. Although epidemiological data are sufficient to link chromium (VI) and lung cancer, experimental data on chromium (III) as a carcinogen are largely negative.

Data from experimental animals on chromium (VI) are considered sufficient and consistent to classify chromium (VI) as a carcinogen in animal bioassays. An inhalation unit risk is

estimated at 1.2×10^{-2} per $\mu g/m^3$ (EPA 1991a) along with an inhalation slope factor of 41 per mg per kg per day (EPA 1992e). Several factors affect the confidence in these values. Assuming a chromium (III) to chromium (VI) ratio of 6:1 may lead to an underestimation of the risk. Using hygiene data from 1949 may result in an overestimation of risk, since it may underestimate true worker exposure. Also, assuming that the smoking habits of chromate workers were similar to those of the general white male population may further overestimate risk, since there are generally more smokers in the industrial field than in the general population (EPA 1991a).

5.3.1.7 Copper

Toxicity Profile

PHYSICOCHEMICAL CHARACTERISTICS							
A. Wt.	MP (°C)	BP (°C)	SG (@25°C)	Water Sol (mg/L)	Log K_{oc}	Log K_{ow}	Vapor Pressure
63.55	1083	2695	8.92	Insoluble	NA	NA	NA

NONCARCINOGENIC TOXICITY VALUES							
Oral RfD (mg/kg/d)		Uncertainty Factor		Inhal. RfC (mg/m^3)		Uncertainty Factor	
Subchron	Chronic	Subchron	Chronic	Subchron	Chronic	Subchron	Chronic
*3.70E-02	*3.70E-02	NA	NA	NA	NA	NA	NA

CARCINOGENIC TOXICITY VALUES					
Cancer Slope Factor (CSF) $(mg/kg/d)^{-1}$		Unit Risk Factor (URF)		Weight-of-Evidence	Reference of study (RfD/RfC/ CSF/URF)
Oral	Inhalation	Oral $(\mu g/L)^{-1}$	Inhalation $(\mu g/m^3)^{-1}$		
NA	NA	NA	NA	D	EPA 1991a; NA; NA; NA

* Calculated from the EPA Office of Drinking Water MCL of 1.3 mg/L by assuming ingestion of 2 liters of water/d by a 70-kg adult.

Copper occurs naturally in elemental form and as a component of many minerals. Because of its high electrical and thermal conductivity, it is widely used in the manufacture of electrical equipment. Common copper salts, such as sulfate, carbonate, cyanide, oxide, and sulfide are used as fungicides, as components of ceramics and pyrotechnics, for electroplating, and for numerous other industrial applications (ACGIH 1986). Copper can be absorbed by the oral, inhalation, and dermal routes of exposure. It is an essential nutrient that is normally present in a wide variety of tissues (ATSDR 1990; EPA 1987).

In humans, ingestion of gram quantities of copper salts may cause gastrointestinal, hepatic, and renal effects with symptoms such as severe abdominal pain, vomiting, diarrhea, hemolysis, hepatic necrosis, hematuria, proteinuria, hypotension, tachycardia, convulsions, coma, and death (USAF 1990). Gastrointestinal disturbances and liver toxicity have also resulted from long-term exposure to drinking water containing 2.2-7.8 mg Cu/L (Mueller-Hoecker et al. 1988; Spitalny et al. 1984). The chronic toxicity of copper has been characterized in patients with Wilson's disease, a genetic disorder causing copper accumulation in tissues. The clinical manifestations of Wilson's disease include cirrhosis of the liver, hemolytic anemia, neurologic abnormalities, and corneal opacities (Goyer 1991; ATSDR 1990; EPA 1987). In animal studies, oral exposure to copper caused hepatic and renal accumulation of copper, liver and kidney necrosis at doses of $\geq$100 mg/kg/d; and hematological effects at doses of 40 mg/kg/d (EPA 1986; Haywood 1985, 1980; Rana and Kumar 1978; Gopinath et al. 1974; Kline et al. 1971).

Acute inhalation exposure to copper dust or fumes at concentrations of 0.075-0.12 mg Cu/m^3 may cause metal fume fever with symptoms such as cough, chills and muscle ache (USAF 1990). Among the reported effects in workers exposed to copper dust are gastrointestinal disturbances, headache, vertigo, drowsiness, and hepatomegaly (Suciu et al. 1981). Vineyard workers chronically exposed to Bordeaux mixture (copper sulfate and lime) exhibit degenerative changes of the lungs and liver. Dermal exposure to copper may cause contact dermatitis in some individuals (ATSDR 1990).

Oral or intravenous administration of copper sulfate increased fetal mortality and developmental abnormalities in experimental animals (Lecyk 1980; Ferm and Hanlon 1974). Evidence also indicates that copper compounds are spermicidal (ATSDR 1990; Battersby et al. 1982).

A Reference Dose (RfD) for elemental copper is not available (EPA 1991b). However, EPA established an action level of 1300 μg/L for drinking water (56 FR 26460, June 7, 1991). Data were insufficient to derive a Reference (RfC) concentration for copper.

No suitable bioassays or epidemiological studies are available to assess the carcinogenicity of copper. Therefore, EPA (1991a,b) has placed copper in weight-of-evidence group D, not classifiable as to human carcinogenicity.

Uncertainty in Toxicity Measures

Noncarcinogenic. EPA currently does not have a chronic health hazard assessment of copper for noncarcinogenic effects. Estimates of subchronic and chronic oral RfDs (both 0.037 mg per kg per day) were derived from the current drinking water standard of 1.3 mg/L (EPA 1991a).

Carcinogenic. No reports are available on copper carcinogenicity in humans. Quantitative estimates of carcinogenic risk for copper by oral and inhalation exposure are not available. Copper is not classified as a human carcinogen (category D) due to the inadequacy of the animal data, equivocal mutagenicity data, and unavailability of human data.

5.3.1.8 Lead

Toxicity Profile

PHYSICOCHEMICAL CHARACTERISTICS							
A. Wt.	MP (°C)	BP (°C)	SG (@25°C)	Water Sol (mg/ℓ)	Log K_{oc}	Log K_{ow}	Vapor Pressure
207.19	327.4	1740	11.35	Insoluble	NA	NA	0.00

NONCARCINOGENIC TOXICITY VALUES							
Oral RfD (mg/kg/d)		Uncertainty Factor		Inhal. RfC (mg/m^3)		Uncertainty Factor	
Subchron	Chronic	Subchron	Chronic	Subchron	Chronic	Subchron	Chronic
NA	NA	NA	NA	NA	NA	NA	NA

CARCINOGENIC TOXICITY VALUES					
Cancer Slope Factor (CSF) $(mg/kg/day)^{-1}$		Unit Risk Factor (URF)		Weight-of-Evidence	Reference of study (RfD/RfC/ CSF/URF)
Oral	Inhalation	Oral $(\mu g/\ell)^{-1}$	Inhalation $(\mu g/m^3)^{-1}$		
NA	NA	NA	NA	B2	NA/NA/ NA/NA

No toxicity values are currently available from EPA for lead exposures. In addition, a BEIA profile is not available for lead. Instead, EPA recommends use of the lead biokinetic model (LEAD 0.60) to estimate blood lead levels in children.

Uncertainty in Toxicity Measures

Noncarcinogenic. Although a great deal of information on the health effects of lead has been obtained through decades of medical observation and scientific research, EPA's RfD work group considered it inappropriate to develop an RfD for inorganic lead. By comparison to most other environmental toxicants, the degree of uncertainty about the health effects of lead is quite low. It appears that some of these effects, particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood lead levels as low as to be essentially without a threshold.

EPA's Office of Solid Waste has established a lead national primary drinking water regulation, which establishes zero as the maximum contaminant level goal and 0.015 mg/L as an action level at the 90th percentile.

Carcinogenic. Available studies lack quantitative exposure information. Further, adjustments for exposures to other metals, such as arsenic, cadmium, and zinc, were not conducted. The cancer excesses observed in the lung and stomach were relatively small (<200). There was no consistency of target tissue sites among the various studies, and no study showed a dose-response relationship. Thus, the available human evidence is considered to be inadequate to refute or demonstrate potential carcinogenicity for humans from lead exposure.

EPA classifies lead as a B2 carcinogen. Weight-of-evidence for such a classification was based on the availability of sufficient evidence from animal studies, where rat bioassays and one mouse assay have shown statistically significant increases in renal tumors with dietary and subcutaneous exposure to several soluble lead salts. Animal assays have provided reproducible results in several laboratories in multiple rat strains with some evidence of multiple tumor sites. Short-term studies show that lead affects gene expression. However, human carcinogenicity evidence is inadequate.

Due to the prevalence of several uncertainties, a quantitative estimate of carcinogenic risk from oral exposure to lead is currently unavailable. Likewise, quantitative estimates of carcinogenic risk from inhalation exposure are unavailable.

5.3.1.9 Manganese

Toxicity Profile

PHYSICOCHEMICAL CHARACTERISTICS							
A. Wt.	MP (°C)	BP (°C)	SG (@25°C)	Water Sol (mg/L)	Log K_{oc}	Log K_{ow}	Vapor Pressure
54.94	1244	2095	7.26	NA	NA	NA	NA

NONCARCINOGENIC TOXICITY VALUES							
Oral RfD (mg/kg/d)		Uncertainty Factor		Inhal. RfC (mg/m^3)		Uncertainty Factor	
Subchron	Chronic	Subchron	Chronic	Subchron	Chronic	Subchron	Chronic
1.00E-01 (water) 1.00E-01 (food)	5.00E-03 (water) 1.40E-01 (food)	1 1	1 1	4.00E-04	4.00E-04	900	300

CARCINOGENIC TOXICITY VALUES					
Cancer Slope Factor (CSF) $(mg/kg/d)^{-1}$		Unit Risk Factor (URF)		Weight-of-Evidence	Reference of study (RfD/RfC/ CSF/URF)
Oral	Inhalation	Oral $(\mu g/L)^{-1}$	Inhalation $(\mu g/m^3)^{-1}$		
NA	NA	NA	NA	D	WHO 1973; Roels et al. 1987;NA; NA

Manganese is an essential trace element in humans, which can elicit a variety of serious toxic responses upon prolonged exposure to elevated concentrations either orally or by inhalation. The central nervous system is the primary target. Initial symptoms are headache, insomnia, disorientation, anxiety, lethargy and memory loss. These symptoms progress with continued exposure and eventually include motor disturbances, tremors and difficulty in walking, similar to symptoms seen in Parkinsonism. These motor difficulties are most often irreversible. Based on human epidemiological studies, 0.8 mg/kg/d for drinking water exposure and 0.34 mg/m^3 in air for inhalation exposure have been estimated as lowest-observed-adverse-effect levels (LOAELs) for central nervous system effects.

Effects on reproduction (decreased fertility, impotence) have been observed in humans with inhalation exposure and in animals with oral exposure at the same or similar doses that initiate the central nervous system effects. An increased incidence of coughs, colds, dyspnea during exercise, bronchitis and altered lung ventilatory parameters have also been seen in humans and animals with inhalation exposure. A possible effect on the immune system may account for some of the respiratory symptoms.

Chronic and oral RfDs of 5E-3 and 1.4E-1 mg/kg/d have been established for manganese exposure via drinking water and food, respectively (EPA 1993). A subchronic oral RfD of 0.10 is listed in HEAST. The oral reference dose (RfD) for food has been calculated by the EPA from a human no-observed-adverse-effect level (NOAEL) of 0.14 mg/kg/d, which was determined from a series of epidemiological studies (Schroeder et al. 1966; WHO 1973; NRC 1989). Large populations with different concentrations of manganese in their drinking water were examined. No adverse effects that were attributable to manganese were seen in any of these groups. The RfD was derived from these studies without uncertainty factors since manganese is essential in human nutrition and the exposure of the most sensitive groups was included in the population examined. Confidence in the oral RfD is rated medium by the EPA.

A reference concentration (RfC) of 0.4 ug/m^3 for both chronic and subchronic inhalation exposure was calculated from a human LOAEL of 0.34 mg/m^3 for respiratory and psychomotor disturbances from an epidemiological study by Roels et al. (1987). The study population was occupationally exposed to airborne manganese dust with a median concentration of 0.97 mg/m^3 for 1 to 19 years with a mean duration of 7.1 years. Neurological examinations, psychomotor tests, lung function tests, blood tests and urine tests were used to determine the possible effects of exposure. The LOAEL was calculated by expanding the dose to continuous exposure. Confidence in the inhalation RfC is rated medium by the EPA.

There are some conflicting data on possible carcinogenesis following injections of manganese chloride and manganese sulfate in mice. However, the EPA weight-of-evidence classification is: D, not classifiable as to human carcinogenicity based on no evidence in humans and inadequate evidence in animals.

Uncertainty in Toxicity Measures

Noncarcinogenic. The information used to determine the chronic oral RfD for manganese in food (1.40E-01 mg/kg/day) was taken from many large populations consuming normal diets over an extended period of time with no adverse health effects. The NOAEL of 10 mg/day (0.14 mg/kg-day for 70 kg adult) for chronic human consumption of manganese in the diet is based on a composite of data from all three references. WHO (1973) reported no adverse effects in humans consuming supplements of 8-9 mg Mn/day (0.11-0.13 mg/kg-day). Schroeder et al. (1966) reported a chronic human NOAEL of 11.5 mg Mn/day (0.16 mg/kg-day) The Food and Nutrition Board of the National Research Council (NRC 1989) determined an "adequate and safe" intake of manganese to be 2-5 mg/day for adults. this level was chosen because it includes an "extra margin of safety" from the level of 10 mg/day, which the NRC considered to be safe for an occasional intake.

The uncertainty factor used in the oral RfD was 1 because the information was taken from many large populations. In addition, manganese is an essential element, being required for normal human growth and maintenance of health. It also has been suggested that children are less susceptible to manganese intoxication and may require slightly higher levels of manganese than adults.

It is emphasized that this oral RfD is based on a total dietary intake of manganese and is not intended to be applied directly to drinking water situation. Because of the greater bioavailability of manganese from water, a separate RfD for water is proposed. The confidence in the critical study can be considered low-to-medium. Confidence in the data base can also be considered medium-to-low. The Greek study is supported by the more severe effects occurring at higher levels in the Japanese study (Kawamura et al. 1941) and the study in rhesus monkeys (Gupta et al. 1980). Overall confidence in the drinking water RfD can be considered medium-to-low.

The RfC for chronic and subchronic inhalation exposure of 0.0004 mg/m^3 was derived from increased prevalence of respiratory symptoms and psychomotor disturbances following occupational exposure to inorganic manganese (Roels et al. 1987). The LOAEL in this study is based on an 8-hour time-weighted average (TWA) occupational exposure. The TWA of total

airborne dust ranged from 0.07 to 8.61 mg/m^3, and the median was 0.97 mg/m^3. This is a respiratory and extrarespiratory effect of a particle exposure.

The uncertainty factor for the chronic RfC is 300, of which 100 reflects 10 to protect sensitive individuals and 10 for use of a LOAEL. An additional factor of 3 was used to account for the less than chronic exposure period. The uncertainty factor for the subchronic RfC (from HEAST) is 900 (EPA 1990b).

The confidence in the principal study (Roels et al. 1987) and the chronic RfC is medium. The LOAEL for respiratory and CNS effects was supported by several other human studies (EPA 1990a). Several limitations preclude assigning a higher confidence to the study. Reflecting medium confidence in the key study and medium confidence in the data base, confidence in the inhalation RfC is medium.

Carcinogenic. Manganese is not classifiable as to human carcinogenicity (weight-of-evidence classification D). No human carcinogenicity data exist and the data available on animal carcinogenicity are inadequate (EPA 1990a).

5.3.1.10 Mercury

Toxicity Profile

PHYSICOCHEMICAL CHARACTERISTICS							
A. Wt.	MP.(°C)	BP (°C)	SG (@25°C)	Water Sol	Log K_{oc}	Log K_{ow}	Vapor Pressure
200.59	-38.87	356.72	13.59	0.28 μmol/L	NA	NA	2.0E-03

NONCARCINOGENIC TOXICITY VALUES							
Oral RfD (mg/kg/d)		Uncertainty Factor		Inhal. RfC (mg/m^3)		Uncertainty Factor	
Subchron	Chronic	Subchron	Chronic	Subchron	Chronic	Subchron	Chronic
3.00E-04	3.00E-04	1000	1000	3.00E-04	3.00E-04	30	30

CARCINOGENIC TOXICITY VALUES					
Cancer Slope Factor (CSF) (mg/kg/d)$^{-1}$		Unit Risk Factor (URF)		Weight-of-Evidence	Reference of study (RfD/RfC/ CSF/URF)
Oral	Inhalation	Oral (μg/L)$^{-1}$	Inhalation (μg/m^3)$^{-1}$		
NA	NA	NA	NA	D	EPA 1991a,b; EPA 1991b; NA; NA

Mercury is a naturally occurring element existing in multiple forms and in various oxidation states. It is used in a wide variety of products and processes. In the environment, mercury may undergo transformations among its various forms and among its oxidation states. Exposure to mercury may occur in both occupational and environmental settings, the latter primarily involving dietary exposure (ATSDR 1989).

Absorption, distribution, metabolism, and excretion of mercury is dependent upon its form and oxidation state (ATSDR 1989; Goyer 1991). Organic mercurials are more readily absorbed than are inorganic forms. An oxidation-reduction cycle is involved in the metabolism of mercury and mercury compounds by both animals and humans (ATSDR 1989). The urine and feces are primary excretory routes. The elimination half-life is 35 to 90 days for elemental mercury and mercury vapor, and about 40 days for inorganic salts (Goyer 1991).

Ingestion of mercury metal is usually without effect (Goldwater 1972). Ingestion of inorganic salts may cause severe gastrointestinal irritation, renal failure and death with acute lethal doses in humans ranging from 1-4 g (ATSDR 1989). Mercuric (divalent) salts are usually more toxic than are mercurous (monovalent) salts (Goyer 1991). Mercury is also known to induce hypersensitivity reactions such as contact dermatitis and acrodynia (pink disease) (Mathesson et al. 1980). Inhalation of mercury vapor may cause irritation of the respiratory tract, renal disorders, CNS effects characterized by neurobehavioral changes, peripheral nervous system toxicity, renal toxicity (immunologic glomerular disease), and death (ATSDR 1989).

Toxicity resulting from subchronic and chronic exposure to mercury and mercury compounds usually involves the kidneys and/or nervous system, the specific target and effect being dependent on the form of mercury (ATSDR 1989). Organic mercury, especially methyl mercury, rapidly enters the central nervous system (CNS) resulting in behavioral and neuromotor disorders (ATSDR 1989; Goyer 1991). The developing CNS is especially sensitive to this effect, as documented by the epidemiologic studies in Japan and Iraq where ingestion of methyl mercury-contaminated food resulted in severe toxicity and death in adults and severe CNS effects in infants (Bakir et al. 1973; AminZaki et al. 1974; Harada et al. 1978). Blood mercury levels of < 10 μg/dL and 300 μg/dL corresponded to mild effects and death, respectively (Bakir et al. 1973). Teratogenic effects due to organic or inorganic mercury exposure do not appear to be well documented for humans or animals, although some evidence exists for mercury-induced menstrual cycle disturbances and spontaneous abortions (Derobert and Tara 1950; Amin-Zaki 1974; ATSDR 1989).

A subchronic and chronic oral RfD of 0.0003 mg/kg/d for methyl mercury is based on the ambient level required to produce a blood mercury level of 200 ng Hg/mL (EPA 1991a,b). A

subchronic and chronic oral RfD of 0.0003 mg/kg/d for inorganic mercury has been proposed and is based on immunologic glomerulonephritis (EPA 1991a,b). A Lowest-Observed-Adverse-Effect-Level (LOAEL) of 0.317 mg Hg/kg/d for inorganic mercury was identified in the key study (Bernaudin et al. 1981). No-Observed-Adverse-Effect-Levels (NOAELs) were not available for oral exposure to inorganic mercury or methyl mercury. A subchronic and chronic inhalation RfC of 0.0003 mg Hg/m^3 for inorganic mercury has been proposed (EPA 1991b) and is based on neurological disorders (increased frequency of intention tremors) following long-term occupational exposure to mercury vapor (Fawer et al. 1983). The LOAELs for subchronic and chronic inhalation exposures to inorganic mercury are 0.32 and 0.03 mg Hg/m^3, respectively. NOAELs were unavailable. An inhalation RfC for methyl mercury has not been determined.

No data were available regarding the carcinogenicity of mercury in humans or animals. The EPA has placed inorganic mercury in weight-of-evidence classification D, not classifiable as to human carcinogenicity. Methyl mercury has not been evaluated by the EPA for evidence of human carcinogenic potential and no carcinogenicity slope factors have been calculated.

Uncertainty in Toxicity Measures

Noncarcinogenic. Noncarcinogenic toxicity values are currently unavailable for mercury in the IRIS data base. However, HEAST lists a tentative subchronic and chronic oral RfD and inhalation RfC of 3×10^{-4} with an associated uncertainty factor of 1000. A great deal of uncertainty surrounds this RfD due to the form of mercury upon which the RfD was based, and due to the studies from which the LOAEL was derived. As part of the BRA for EFPC, the toxicity of mercury was explored further. A critical review of the literature was conducted along with recent data on mercury toxicity in laboratory test species. Alternate RfDs were derived for mercury species likely to be found in flood-plain soils of EFPC. This discussion is presented in Sect. 5.3.2 and in greater detail in Appendix N.

Carcinogenic. EPA does not classify mercury as a carcinogen (category D) based on the unavailability of human data and inadequacy of experimental data.

5.3.1.11 Nickel

Toxicity Profile

PHYSICOCHEMICAL CHARACTERISTICS							
A. Wt.	MP (°C)	BP (°C)	SG (@25°C)	Water Sol (mg/L)	Log K_{oc}	Log K_{ow}	Vapor Pressure
58.69	1550	2837	8.90	Insoluble	NA	NA	0.00

NONCARCINOGENIC TOXICITY VALUES							
Oral RfD (mg/kg/d)		Uncertainty Factor		Inhal. RfC (mg/m^3)		Uncertainty Factor	
Subchron	Chronic	Subchron	Chronic	Subchron	Chronic	Subchron	Chronic
2.00E-02	2.00E-02	300	300	NA	NA	NA	NA

CARCINOGENIC TOXICITY VALUES					
Cancer Slope Factor (CSF) $(mg/kg/d)^{-1}$		Unit Risk Factor (URF)		Weight-of-Evidence	Reference of study (RfD/RfC/ CSF/URF)
Oral	Inhalation	Oral $(\mu g/L)^{-1}$	Inhalation $(\mu g/m^3)^{-1}$		
NA	8.40E-01 (refin. dust) 1.70E+00 (subsulfide)	NA	2.40E-04 (refin. dust) 4.80E-04 (subsulfide)	A (refin.dust) A (subsulfide)	Ambrose et al. 1976; NA; EPA 1991a

Nickel is a naturally occurring element that may exist in various mineral forms. Nickel is used in a wide variety of applications including metallurgical processes and electrical components, such as batteries (ATSDR 1988; USAF 1990). There is some evidence that nickel may be an essential trace element for mammals.

The absorption of nickel is dependent on its physicochemical form, with water soluble forms being more readily absorbed. The metabolism of nickel involves conversion to various chemical forms and binding to various ligands (ATSDR 1988). Nickel is excreted in the urine and feces with relative amounts for each route being dependent on the route of exposure and chemical form. Most nickel enters the body via food and water consumption, although inhalation exposure in occupational settings is a primary route for nickel-induced toxicity.

In large doses (>0.5 g) some forms of nickel may be acutely toxic to humans when taken orally (Daldrup et al. 1983; Sunderman et al. 1988). Oral LD_{50} values for rats range from 67 mg Ni/kg (nickel sulfate hexahydrate) to > 9000 mg Ni/kg (nickel powder) (ATSDR 1988). Toxic effects of oral exposure to nickel usually involve the kidneys with some evidence from animal studies showing a possible developmental/reproductive toxicity effect (ATSDR 1988; Goyer 1991).

Inhalation exposure to some nickel compounds will cause toxic effects in the respiratory tract and immune system (ATSDR 1988; Goyer 1991). Inhalation LC_{50} values for animals range from 0.97 mg Ni/m^3 for rats (6-hour exposure) to 15 mg Ni/m^3 for guinea pigs (time not specified) (USAF 1990). Acute inhalation exposure of humans to nickel may produce headache, nausea, respiratory disorders and death (Goyer 1991). Asthmatic conditions have also been documented for inhalation exposure to nickel (Goyer 1991). Soluble nickel compounds tend to be more toxic than insoluble compounds (Goyer 1991). Nickel carbonyl is known to be extremely toxic to humans upon acute inhalation exposure (Goyer 1991).

Sensitivity reactions to nickel are well documented and usually involve contact dermatitis reactions resulting from contact with nickel-containing items such as cooking utensils, jewelry, coins, etc (ATSDR 1988).

A chronic and subchronic oral RfD of 0.02 mg/kg/d for soluble nickel salts is based on changes in organ and body weights of rats receiving dietary nickel sulfate hexahydrate (5 mg/kg/d) for two years (EPA 1991a). A No-Observed-Adverse-Effects-Level (NOAEL) and Lowest-Observed-Adverse-Effects-Level (LOAEL) of 5 mg/kg/day and 50 mg/kg/d, respectively, were reported in the key study (Ambrose et al. 1976). An uncertainty factor of 300 reflects interspecies extrapolation uncertainty, protection of sensitive populations, and a modifying factor of 3 for a database deficient in reproductive/developmental studies. An inhalation RfC for soluble nickel salts is under review and not currently available.

The primary target organs for nickel-induced systemic toxicity are the lungs and upper respiratory tract for inhalation exposure and the kidneys for oral exposure (ATSDR 1988; Goyer 1991). Other target organs include the cardiovascular system, immune system and the blood.

Epidemiologic studies have shown that occupational inhalation exposure to nickel dust (primarily nickel subsulfide) at refineries has resulted in increased incidences of pulmonary and nasal cancer (NAS 1975; Enterline and Marsh 1982; ATSDR 1988). Inhalation studies using rats have also shown nickel subsulfide or nickel carbonyl to be carcinogenic (Sunderman et al. 1959; Sunderman and Donnelly 1965; Ottolenghi et al. 1974). Based on these data, the EPA has classified nickel subsulfide and nickel refinery dust in weight-of-evidence group A, Human Carcinogen. Carcinogenicity slope factors of 1.7E+0 and 8.4E-01 $(mg/kg/d)^{-1}$ and unit risks of 4.8×10^{-4} $(\mu g/m^3)^{-1}$ and 2.4×10^{-4} $(\mu g/m^3)^{-1}$ have been calculated for nickel subsulfide and nickel refinery dust, respectively (EPA 1991a). Based on an increased incidence of pulmonary carcinomas and malignant tumors in animals exposed to nickel carbonyl by inhalation or by intravenous injection, this compound had been placed in weight-of-evidence group B2, probable human carcinogen. No unit risk values were available for nickel carbonyl.

Uncertainty in Toxicity Measures

Noncarcinogenic. The RfD for chronic and subchronic oral exposure for nickel was estimated at 0.02 mg per kg per day based on decreased body and organ weights in a chronic feeding study on rats (Ambrose et al. 1976). The NOAEL and LOAEL for this study were 100 and 1000 ppm, respectively (converted to 5 and 50 mg per kg per day, respectively; assuming 1 ppm = 0.05 mg per kg per day).

An uncertainty factor of 300 was used to derive the RfD: 10 for interspecies extrapolation and 10 to protect sensitive populations, and an additional factor of 3 was used because of inadequacies in several reproductive studies (RTI 1987; Ambrose et al. 1976). During the gestation and postnatal development of $F1_b$ litters in the RTI (1987) study, temperatures were approximately 10°F higher than normal at certain times, which makes evaluation of this part of the reproductive study impossible. In the Ambrose et al. (1976) study, there were some statistical design limitations, such as small sample size and use of pups rather than litters as the unit for comparison. The modifying factor for the oral RfD is one.

The chronic study (Ambrose et al. 1976) was properly designed and provided adequate toxicological endpoints; however, high mortality occurred in the controls (44/50). Therefore, a low confidence is recommended for the study. The data base provided adequate supporting subchronic studies, one by gavage and the other in drinking water. A medium confidence level in the data base is recommended, since inadequacies exist in the remaining reproduction data.

Carcinogenic. EPA has not evaluated soluble salts of nickel as a class of compounds for potential human carcinogenicity. This is largely due to the inconclusive nature or inadequacy of the available experimental and epidemiological data (EPA 1991a). However, nickel refinery dust and specific nickel compounds (i.e., nickel carbonyl and nickel subsulfide) have been evaluated. Carcinogenicity slope factors of 1.7 and 0.84 $(mg/kg/d)^{-1}$ and unit risks of 4.8×10^{-4} and 2.4×10^{-4} $(\mu g/m^3)^{-1}$ have been calculated for nickel subsulfide and nickel refinery dust, respectively (EPA 1991a). Heath hazard assessments on nickel and its compounds for varied exposure durations are currently unavailable.

5.3.1.12 Silver

Toxicity Profile

PHYSICOCHEMICAL CHARACTERISTICS							
A. Wt.	MP (°C)	BP (°C)	SG (@25°C)	Water Sol (mg/L)	Log K_{oc}	Log K_{ow}	Vapor Pressure
107.87	960.5	2000	10.49	Insoluble	NA	NA	0.00
NONCARCINOGENIC TOXICITY VALUES							
Oral RfD (mg/kg/d)		Uncertainty Factor		Inhal. RfC (mg/m³)		Uncertainty Factor	
Subchron	Chronic	Subchron	Chronic	Subchron	Chronic	Subchron	Chronic
5.00E-03	5.00E-03	3	3	NA	NA	NA	NA
CARCINOGENIC TOXICITY VALUES							
Cancer Slope Factor (CSF) (mg/kg/d)$^{-1}$			Unit Risk Factor (URF)			Weight-of-Evidence	Reference of study (RfD/RfC/ CSF/URF)
Oral		Inhalation	Oral (μg/L)$^{-1}$		Inhalation (μg/m³)$^{-1}$		
NA		NA	NA		NA	D	Gaul & Staud 1935; NA; NA; NA

Silver is a relatively rare metal that occurs naturally in the earth's crust and is released to the environment from various industrial sources. Human exposure to silver and silver compounds can occur orally, dermally, or by inhalation. Silver is found in most tissues, but has no known physiologic function.

In humans, accidental or intentional ingestion of large doses of silver nitrate has produced corrosive damage of the gastrointestinal tract, abdominal pain, diarrhea, vomiting, shock, convulsions, and death (EPA 1985). Respiratory irritation was noted following acute inhalation exposure to silver or silver compounds. Silver nitrate solutions are highly irritating to the skin, mucous membranes, and eyes (Stokinger 1981).

Ingestion, inhalation, or dermal absorption of silver may cause argyria, the most common indicator of long-term exposure to silver or silver compounds in humans. Argyria is a gray or blue-gray, permanent discoloration of the skin and mucous membranes that is not a toxic effect per se, but is considered cosmetically disfiguring. Chronic inhalation exposure of workers to silver oxide and silver nitrate dusts resulted in upper and lower respiratory irritation, deposition of granular silver-containing deposits in the eyes, impaired night vision, and abdominal pain (Rosenman et al. 1979). Mild allergic responses have been attributed to dermal contact with silver (ATSDR 1990).

In long-term oral studies with experimental animals, silver compounds have produced slight thickening of the basement membranes of the renal glomeruli, growth depression, shortened lifespan, and granular silver-containing deposits in skin, eyes, and internal organs (Matuk et al. 1981; Olcott 1948, 1950). Hypoactivity was seen in rats subchronically exposed to silver nitrate in drinking water (Rungby and Danscher 1984).

A reference dose for oral exposure is estimated as 0.005 mg/kg/d based on the observation of argyria in a 2 to 9-year human intravenous study (EPA 1991b). Data are presently insufficient to derive a Reference Concentration (RfC) for silver (EPA 1991b).

Data adequate for evaluating the carcinogenicity of silver to humans or animals by ingestion, inhalation, or other routes of exposure were not found. Based on EPA guidelines, silver is placed in weight-of-evidence group D, not classifiable as to human carcinogenicity (EPA 1991b).

Uncertainty in Toxicity Measures

Noncarcinogenic. An RfD for chronic and subchronic oral exposure for silver is estimated as 0.005 mg per kg per day based on the observation of argyria in a 2- to 9-year human intravenous study (Gaul and Staud 1935). Argyria is a medially benign but permanent bluish-gray discoloration of the skin and conjunctiva of the eye, and in some severe cases even blackening of the skin with a metallic luster. Argyria results from the deposition of silver in the dermis and also from silver-induced production of melanin. The LOAEL for this study was 1 g (total dose) converted to an oral dose of 0.014 mg per kg per day. This dose was based on conversion from the total i.v. dose to a total oral dose of 25 g (i.v. dose of 1 g divided by 0.04, assumed oral retention factor) and dividing by 70 kg (adult body weight) and 25,500 days (a lifetime, or 70 years).

An uncertainty factor of 3 is applied to account for minimal effects in a subpopulation that has exhibited an increased propensity for the development of argyria. The oral RfD modifying factor is one.

Confidence in the RfD can be considered low-to-medium because, while the critical effect has been demonstrated in humans following oral administration of silver, the quantitative risk estimate is based on a study utilizing intravenous administration, and thus necessitates a dose conversion with inherent uncertainties (EPA 1991b). Confidence in the data base is low because the studies were not controlled. The critical human study is considered to be of medium confidence.

Carcinogenic. Only equivocal evidence exists to suggest that silver has carcinogenic activity in experimental animals. Silver implants and injected colloidal suspensions are reported to produce tumors or hyperplasia at the site of application in several studies (Furst 1981; Schmahl and Steinhoff 1960). However, it is suggested that the effects are due to the physical form of the metal or to its action as an exogenous irritant. No studies suggest that silver is carcinogenic in humans.

EPA has not classified silver as to human carcinogenicity (weight-of-evidence category D). This is based upon the ambiguity of data on implantation-induced carcinogenesis and the negative results obtained in studies employing colloidal and powder forms of silver (EPA 1991b).

5.3.1.13 Vanadium

Toxicity Profile

PHYSICOCHEMICAL CHARACTERISTICS							
A. Wt.	MP (°C)	BP (°C)	SG (@25°C)	Water Sol (mg/L)	Log K_{oc}	Log K_{ow}	Vapor Pressure
50.94	1917	3380	6.11	Insoluble	NA	NA	NA

NONCARCINOGENIC TOXICITY VALUES							
Oral RfD (mg/kg/d)		Uncertainty Factor		Inhal. RfC (mg/m^3)		Uncertainty Factor	
Subchron	Chronic	Subchron	Chronic	Subchron	Chronic	Subchron	Chronic
7.00E-03	7.00E-03	100	100	NA	NA	NA	NA

CARCINOGENIC TOXICITY VALUES					
Cancer Slope Factor (CSF) $(mg/kg/d)^{-1}$		Unit Risk Factor (URF)		Weight-of-Evidence	Reference of study (RfD/RfC/ CSF/URF)
Oral	Inhalation	Oral $(\mu g/L)^{-1}$	Inhalation $(\mu g/m^3)^{-1}$		
NA	NA	NA	NA	NA	Schroeder et al. 1970; NA; NA; NA

Vanadium is a metallic element that occurs in six oxidation states and numerous inorganic compounds. Some of the more important compounds are vanadium pentoxide (V_2O_5), sodium metavanadate ($NaVO_3$), sodium orthovanadate (Na_3VO_4), vanadyl sulfate ($VOSO_4$), and ammonium vanadate (NH_4VO_3). Vanadium is used primarily as an alloying agent in steels and nonferrous metals (ATSDR 1990). Vanadium compounds are also used as catalysts and in chemical, ceramic or specialty applications.

Vanadium compounds are poorly absorbed through the gastrointestinal system (0.5-2% of dietary amount) (NRCC 1980; ICRP 1960; Byrne and Kosta 1978), but slightly more readily absorbed through the lungs (20-25%) (ICRP 1960; Davies and Bennett 1983). Absorbed vanadium is widely distributed in the body, but short-term localization occurs primarily in bone, kidneys, and liver (Vouk 1979; Roshchin et al. 1980; Parker et al. 1980; Sharma et al. 1980; Wiegmann et al. 1982). In the body, vanadium can undergo changes in oxidation state (interconversion of vanadyl (+4) and vanadate (+5) forms) and it can also bind with blood protein (transferrin) (Harris et al. 1984). Vanadium is excreted primarily in the feces following oral exposures and primarily in the urine following inhalation exposures (Tipton et al. 1969; ATSDR 1990).

The toxicity of vanadium depends on its physicochemical state; particularly on its valence state and solubility. Based on acute toxicity, pentavalent NH_4VO_3 has been reported to be more than twice as toxic as trivalent VCl_3 and more than 6 times as toxic as divalent VI_2. Pentavalent V_2O_5 has been reported to be more than 5 times as toxic as trivalent V_2O_3 (Roschin 1967). In animals, acutely toxic oral doses cause vasoconstriction, diffuse desquamative enteritis, congestion and fatty degeneration of the liver, congestion and focal hemorrhages in the lungs and adrenal cortex (Gosselin et al. 1984). Minimal effects seen after subchronic oral exposures to animals include diarrhea, altered renal function, and decreases in erythrocyte counts, hemoglobin, and hematocrit (Domingo et al. 1985; Zaporowska and Wasilewski 1990). In humans, intestinal cramps and diarrhea may occur following subchronic oral exposures. These studies indicate that for subchronic and chronic oral exposures the primary targets are the digestive system, kidneys, and blood.

Reference Doses (RfD) for chronic oral exposures are: 0.007 mg/kg/d for vanadium; 0.009 mg/kg/day for vanadium pentoxide; 0.02 mg/kg/d for vanadyl sulfate; and 0.001 mg/kg/d for sodium metavanadate (EPA 1987, 1991a,b). The subchronic RfDs for these compounds are the same as the chronic RfDs, except for sodium metavanadate, which is 0.01 mg/kg/day (EPA 1987, 1991a,b).

Inhalation exposures to vanadium and vanadium compounds result primarily in adverse effects to the respiratory system (Sax 1984; ATSDR 1990). In laboratory studies, minimal effects (throat irritation and coughing) occurred after an 8-hr exposure to 0.1 mg V/m^3 (Zenz and Berg 1967). In studies on workers occupationally exposed to vanadium, the most common reported symptoms were: irritation of the respiratory tract, conjunctivitis, dermatitis, cough, bronchospasm, pulmonary congestion, and bronchitis (Symanski 1939; Sjoberg 1950, 1951, 1955, 1956; Vintinner et al. 1955; Lewis 1959; Tebrock and Machle 1968; Roshchin 1968; Kiviluoto et al. 1981b). Quantitative data are, however, insufficient to derive a subchronic or chronic inhalation Reference Concentration (RfC) for vanadium or vanadium compounds.

There is little evidence that vanadium or vanadium compounds are reproductive toxins or teratogens. There is also no evidence that any vanadium compound is carcinogenic; however, very few adequate studies are available for evaluation. Vanadium has not been classified as to carcinogenicity by the EPA (1991a).

Uncertainty in Toxicity Measures

Noncarcinogenic. While the IRIS data base states that no data are available to derive a chronic oral RfD, the HEAST document (EPA 1991a) presents a chronic and subchronic oral RfD of 0.007 mg per kg per day. This estimate is based on a NOAEL of 5 ppm (0.7 mg per kg per day) vanadium from vanadyl sulfate in drinking water in a rat lifetime study (Schroeder et al. 1970). The uncertainty factor for the RfD estimate was 100.

Carcinogenic. No data are available to suggest that vanadium has carcinogenic or mutagenic effects in humans or experimental animals.

5.3.1.14 Zinc

Toxicity Profile

PHYSICOCHEMICAL CHARACTERISTICS							
A. Wt.	MP (°C)	BP (°C)	SG (@25°C)	Water Sol (mg/L)	Log K_{oc}	Log K_{ow}	Vapor Pressure
65.38	419.5	908	7.14	Insoluble	NA	NA	0.00

NONCARCINOGENIC TOXICITY VALUES							
Oral RfD (mg/kg/d)		Uncertainty Factor		Inhal. RfC (mg/m^3)		Uncertainty Factor	
Subchron	Chronic	Subchron	Chronic	Subchron	Chronic	Subchron	Chronic
3.00E-01	3.00E-01	3	3	NA	NA	NA	NA

CARCINOGENIC TOXICITY VALUES					
Cancer Slope Factor (CSF) $(mg/kg/d)^{-1}$		Unit Risk Factor (URF)		Weight-of-Evidence	Reference of study (RfD/RfC/ CSF/URF)
Oral	Inhalation	Oral $(\mu g/L)^{-1}$	Inhalation $(\mu g/m^3)^{-1}$		
NA	NA	NA	NA	D	EPA 1991a, 1992; NA; NA; NA

Zinc is used primarily in galvanized metals and metal alloys, but zinc compounds also have wide commercial applications as chemical intermediates, catalysts, pigments, vulcanization activators and accelerators in the rubber industry, UV stabilizers, and supplements in animal feeds and fertilizers. They are also used in rayon manufacture, smoke bombs, soldering fluxes, mordants for printing and dyeing, wood preservatives, mildew inhibitors, deodorants, antiseptics, and astringents (Lloyd 1984; ATSDR 1989). In addition, zinc phosphide is used as a rodenticide.

Zinc is an essential element with recommended daily allowances ranging from 5 mg for infants to 15 mg for adult males (NRC 1989).

Gastrointestinal absorption of zinc is variable (20-80%) and depends on the chemical compound as well as on zinc levels in the body and dietary concentrations of other nutrients (EPA 1984). In individuals with normal zinc levels in the body, gastrointestinal absorption is 20-30% (ATSDR 1989). Information on pulmonary absorption is limited and complicated by the potential for gastrointestinal absorption due to mucociliary clearance from the respiratory tract and subsequent swallowing. Zinc is present in all tissues with the highest concentrations in the prostate, kidney, liver, heart, and pancreas. Zinc is a vital component of many metalloenzymes such as carbonic anhydrase, which regulates CO_2 exchange (Stokinger 1981). Homeostatic mechanisms involving metallothionein in the mucosal cells of the gastrointestinal tract regulate zinc absorption and excretion (ATSDR 1989).

In humans, acutely toxic oral doses of zinc cause nausea, vomiting, diarrhea, and abdominal cramps and in some cases gastric bleeding (Elinder 1986; Moore 1978; ATSDR 1989). Ingestion of zinc chloride can cause burning in the mouth and throat, vomiting, pharyngitis, esophagitis, hypocalcemia, and elevated amylase activity indicative of pancreatitis (Chobanian 1981). Zinc phosphide, which releases phosphine gas under acidic conditions in the stomach, can cause vomiting, anorexia, abdominal pain, lethargy, hypotension, cardiac arrhythmias, circulatory collapse, pulmonary edema, seizures, renal damage, leukopenia, and coma and death in days to weeks (Mack 1989). The estimated fatal dose is 40 mg/kg. Animals dosed orally with zinc compounds develop pancreatitis, gastrointestinal and hepatic lesions, and diffuse nephrosis.

Gastrointestinal upset has also been reported in individuals taking daily dietary zinc supplements for up to 6 weeks (Samman and Roberts 1987). There is also limited evidence that the human immune system may be impaired by subchronic exposures (Chandra 1984). In animals, gastrointestinal and hepatic lesions, (Allen et al. 1983; Brink et al. 1959); pancreatic lesions (Maita et al. 1981; Drinker et al. 1927a); anemia (ATSDR 1989; Fox and Jacobs 1986; Maita et al. 1981); and diffuse nephrosis (Maita et al. 1981; Allen et al. 1983) have been observed following subchronic oral exposures.

Chronic oral exposures to zinc have resulted in hypochromic microcytic anemia associated with hypoceruloplasminemia, hypocupremia, and neutropenia in some individuals (Prasad et al. 1978; Porter et al. 1977). Anemia and pancreatitis were the major adverse effects observed in chronic animal studies (Aughey et al. 1977; Drinker et al. 1927a; Walters and Roe 1965; Sutton and Nelson 1937). Teratogenic effects have not been seen in animals exposed to zinc; however, high oral doses can affect reproduction and fetal growth (Ketcheson et al. 1969; Schlicker and Cox 1967 1968; Sutton and Nelson 1937).

The reference dose for chronic oral exposure to zinc is 0.3 mg/kg/d based on clinical data from the investigation of effects of oral zinc supplements on copper and iron balance (IRIS Database, Oct. 1991). The currently accepted RfD for subchronic exposures is also 0.3 mg/kg/d based on clinical data demonstrating zinc-induced copper deficiency and anemia in patients taking zinc sulfate for the treatment of sickle cell anemia (EPA 1992). The chronic oral RfD for zinc phosphide is 0.0003 mg/kg/d (EPA 1991a), and the subchronic RfD is 0.003 mg/kg/d (EPA 1992).

Under occupational exposure conditions, inhalation of zinc compounds (mainly zinc oxide fumes) can result in a condition identified as "metal fume fever," which is characterized by nasal passage irritation, cough, rales, headache, altered taste, fever, weakness, hyperpnea, sweating, pains in the legs and chest, leukocytosis, reduced lung volume, and decreased diffusing capacity of carbon monoxide (ATSDR 1989; Bertholf 1988). Inhalation of zinc chloride can result in nose and throat irritation, dyspnea, cough, chest pain, headache, fever, nausea and vomiting, and respiratory disorders such as pneumonitis and pulmonary fibrosis (ITII 1988; ATSDR 1989; Nemery 1990). Pulmonary inflammation and changes in lung function have also been observed in inhalation studies on animals (Amur et al. 1982; Lam et al. 1985; Drinker and Drinker 1928).

Although "metal fume fever" occurs in occupationally exposed workers, it is primarily an acute and reversible effect that is unlikely to occur under chronic exposure conditions when zinc air concentrations are less than 8-12 mg/m^3 (ATSDR 1989). Gastrointestinal distress, as well as enzyme changes indicative of liver dysfunction, have also been reported in workers occupationally exposed to zinc (NRC 1979; Stokinger 1981; EPA 1991a; Guja 1973; Badawy et al. 1987a); however, it is unclear as to what extent these effects might have been caused by pulmonary clearance, and subsequent gastrointestinal absorption. Consequently, there are no clearly defined toxic effects that can be identified as resulting specifically from pulmonary absorption following chronic low level inhalation exposures. Animal data for chronic inhalation exposures are not available.

An inhalation reference concentration has not been derived for zinc or zinc compounds (EPA 1992).

No case studies or epidemiologic evidence has been presented to suggest that zinc is carcinogenic in humans by the oral or inhalation route (EPA 1991a). In animal studies, zinc sulfate in drinking water or zinc oleate in the diet of mice for a period of one year did not result in a statistically significant increase in hepatomas, malignant lymphomas, or lung adenomas (Walters and Roe 1965); however, in a 3-year, 5-generation study on tumor-resistant and tumor-susceptible strains of mice, exposure to zinc in drinking water resulted in increased frequencies of tumors from the F_0 to the F_4 generation in the tumor-resistant strain (from 0.8 to 25.7%, vs. 0.0004% in the controls), and higher tumor frequencies in two tumor-susceptible strains (43.4% and 32.4% vs. 15% in the controls) (Halme 1961).

Zinc is placed in weight-of-evidence Group D, not classifiable as to human carcinogenicity due to inadequate evidence in humans and animals (U.S. EPA 1991a).

Uncertainty in Toxicity Measures

Noncarcinogenic. The chronic oral RfD of 0.3 mg per kg per day was based on a 47% decrease in erythrocyte superoxide dismutase concentration in adult human females after 10 weeks of zinc exposure. The LOAEL for this study was 1.0 mg per kg per day. A subchronic oral RfD of 0.2 mg per kg per day is presented in HEAST.

An uncertainty factor of 3 was used for the chronic RfD, based on a minimal LOAEL from a moderate-duration study of the most sensitive humans and consideration of a substance that is an essential dietary nutrient (EPA 1991a). The uncertainty factor for the subchronic RfD is also 3 (EPA 1992e).

The level of confidence in the studies was medium because they are all well-conducted clinical studies with many biochemical parameters investigated, but only a few humans were tested. The confidence in the overall data base was medium because these studies are all of short duration. Medium confidence in the RfD follows (EPA 1991a).

Carcinogenic. Zinc and its compounds are not classifiable as to human carcinogens (weight-of-evidence classification D) based on inadequate evidence in humans and animals (EPA 1991a). No reports are available on the possible carcinogenicity of zinc and compounds per se in humans. Case studies have been used to evaluate the effects of zinc administered for therapeutic reasons (Porter et al. 1977; Prasad et al. 1978).

5.3.1.15 Polycyclic Aromatic Hydrocarbons (PAHs)

Toxicity Profile

CAS Numbers:

Benzo(a)anthracene [B(a)A]:	56-55-3	Benzo(b)fluoranthene [B(b)F]:	205-99-2
Benzo(k)fluoranthene [B(k)F]:	207-08-9	Benzo(a)pyrene [B(a)P]:	50-32-8
Chrysene (C):	218-01-9	Dibenzo(a,h)anthracene (DBA):	53-70-3
Indeno(1,2,3-cd)pyrene (IP):	193-39-5	Pyrene (P):	129-00-0

PHYSICOCHEMICAL CHARACTERISTICS

A. Wt.	MP (°C)	BP (°C)	SG (@25°C)	Water Sol (mg/L)	Log K_{ow}	Log K_{oc}	Vapor Pressure
B[a]A: 228.30 B[b]F: 252.32 B[k]F: 252.32 B[a]P: 252.32 C: 228.30 DBA: 278.36 IP: 276.34 P: 202.26	B[a]A: 162 B[b]F: 168 B[k]F: 217 B[a]P: 179 C: 255 DBA: 269 IP: 160 P: 156	B[a]A: 437.6 B[b]F: ND B[k]F: 480 B[a]P: 495 C: 448 DBA: 524 IP: 536 P: 393	B[a]A: 1.274 B[b]F: ND B[k]F: ND B[a]P: 1.351 C: 1.274 DBA: 1.282 IP: ND P: 1.271	B[a]A: 0.014 B[b]F: 0.0012 B[k]F: 5.5E-4 B[a]P: 0.0038 C: 0.006 DBA: 0.0005 IP: 0.062 P: 0.013	B[a]A: 6.14 B[b]F: 5.74 B[k]F: 6.64 B[a]P: 5.60 C: 5.39 DBA: 6.22 IP: 7.49 P: 4.66	B[a]A: 5.61 B[b]F: 6.57 B[k]F: 6.85 B[a]P: 5.99 C: 5.60 DBA: 6.36 IP: 7.70 P: 4.88	B[a]A: 5E-9 B[b]F: 5E-7 B[k]F:9E-11 B[a]P: 5E-9 C: 6.3E-9 DBA:1E-11 IP: 10E-10 P: 6.85E-7

NONCARCINOGENIC TOXICITY VALUES

[a]Oral RfD (mg/kg/d)		Uncertainty Factor		Inhal. RfC (mg/m³)		Uncertainty Factor	
Subchron	Chronic	Subchron	Chronic	Subchron	Chronic	Subchron	Chronic
B[a]A: 3.00E-01 B[b]F: 3.00E-01 B[k]F: 3.00E-01 B[a]P: 3.00E-01 C: 3.00E-01 DBA: 3.00E-01 IP: 3.00E-01 P: 3.00E-01	B[a]A: 3.00E-02 B[b]F: 3.00E-02 B[k]F: 3.00E-02 B[a]P: 3.00E-02 C: 3.00E-02 DBA: 3.00E-02 IP: 3.00E-02 P: 3.00E-02	P: 300	P: 3000	NA	NA	NA	NA

CARCINOGENIC TOXICITY VALUES

[b]Cancer Slope Factor (CSF) (mg/kg/d)⁻¹		Unit Risk Factor (URF)		Weight-of-Evidence	Reference of study (RfD/RfC/ CSF/URF)
Oral	Inhalation	Oral (µg/L)⁻¹	Inhalation (µg/m³)⁻¹		
B[a]A: 7.30E-01 B[b]F: 7.30E-01 B[k]F: 7.30E-01 B[a]P: 7.30E+00 C: 7.30E-02 DBA: 7.30E+00 IP: 7.30E-01	B[a]A:6.10E-01 B[b]F:6.10E-01 B[k]F:6.10E-01 B[a]P: 6.10E+00 C:6.10E-02 DBA: 6.10E+00 IP: 6.10E-01	B[a]A: NA B[b]F: NA B[k]F: NA B[a]P: 2.10E-04 C: NA DBA: NA IP: NA	B[a]A: NA B[b]F: NA B[k]F: NA B[a]P:1.70E-03 C: NA DBA: NA IP: NA	B[a]A: B2 B[b]F: B2 B[k]F: B2 B[a]P: B2 C: B2 DBA: B2 IP: B2	EPA 1989; NA; Thyssen et al. 1981; Neal and Rigdon 1967

[a] In the absence of toxicity data, the RfDs for pyrene have been adopted for these compounds.

[b] The CSFs for these compounds have been estimated by multiplying the CSF for benzo(a)pyrene by a toxicity equivalence factor (EPA 1992a, Region IV Interim Guidance).

PAHs are a diverse class of compounds consisting of substituted and unsubstituted polycyclic and heterocyclic aromatic rings formed as a result of incomplete combustion of organic compounds with insufficient oxygen (EPA 1980).

General Toxicity. Data on the health effects of PAHs predominantly concern their possible carcinogenic effects. However, application of carcinogenic PAHs on the skin in experimental animals is reported to cause destruction of sebaceous glands (skin), hyperkeratosis, hyperplasia, and ulceration. Many carcinogenic PAHs are immunosuppressive agents. Toxic manifestations in the liver and kidney are reported in experimental animals exposed to PAHs. Workers exposed to PAH-containing materials have exhibited chronic dermatitis, hyperkeratosis, and other skin manifestations (EPA 1980).

There is an increasing body of evidence that several carcinogenic PAHs produce severe, long-term immunotoxicity. Exposure to benzo(a)pyrene results in a long-lasting reduction in the activity of antibody-producing cells. Exposure of pregnant mice to a single dose (100 to 150 mg/kg) of benzo(a)pyrene gave severe suppression of antibody response to pups shortly after birth (Dean et al. 1986).

A chronic oral RfD of 0.03 mg/(kg • d) was estimated for pyrene based on kidney effects (renal tubular pathology and decreased kidney weights) in a mouse subchronic oral bioassay (EPA 1989g). A subchronic toxicity value of 0.3 mg/(kg • d) is listed in HEAST. In the absence of specific toxicity data for noncarcinogenic effects for the PAHs that are COCs in this assessment, the RfDs for pyrene have been adopted.

Evidence of Carcinogenicity. Several PAHs are strongly implicated in the proven association between smoking and lung cancer, between occupational exposure to coke-oven emissions, coal-tar pitch, mineral oils, and similar products and cancer of skin, lung, bladder, and the gastro-intestinal tract. However, in humans, because of the fact that exposure involves complex mixtures where, clearly, there is interaction between a number of initiators and promoters, it is more difficult to assess the activity of individual members of the group (EPA 1980).

A number of PAHs have been found to induce tumors in experimental animals upon exposure by different routes of administration [B(a)A, B(b)F, B(k)F, B(a)P, DBA, and IP]. The more active congeners, like dibenzo(a,h)anthracene, induce tumors in multiple sites, and also act as transplacental carcinogens. For benzo(a)pyrene, lung and liver tumors have been induced by exposure to pregnant mice (Dean et al. 1986). There is limited evidence that chrysene is carcinogenic in animals, and the available data for pyrene provide no evidence that it is carcinogenic.

The skin of many rodent species, like the mouse, is extremely sensitive to chemical carcinogens of this type, and experiences from epidemiological investigations tend to demonstrate a much lower sensitivity of human skin than that of the mouse, a finding that has been corroborated by investigations in monkeys (Pucknat 1981).

In the past, estimation of risk from carcinogenic PAHs has been conducted assuming that all carcinogenic PAHs have the same potency as benzo(a)pyrene. This approach has little scientific validity and may result in great overestimation of risk. In this BRA, as specified in the EPA Region IV Guidance, toxicity equivalence factors (TEFs) have been used to obtain toxicity values relative to benzo(a)pyrene (the latter of which is assigned a TEF of one). This relative potency approach and further specifics are described in greater detail in Sect. 5.3.3. The toxicity values listed in this section incorporate the TEFs specified in the EPA Region IV Guidance. The carcinogenic effects of the individual PAHs are summarized below.

Benzo(a)anthracene. Using EPA Region IV TEFs, the oral and inhalation slope factors estimated for benzo(a)anthracene are 0.73 and 0.61 $[mg/(kg \cdot d)]^{-1}$, respectively. Benzo(a)-anthracene is classified as a probable human carcinogen (weight-of-evidence classification B2).

Benzo(b)fluoranthene. The oral and inhalation slope factors for benzo(b)fluoranthene are 0.73 and 0.61 $[mg/(kg \cdot d)]^{-1}$, respectively, using the EPA Region IV TEFs. This compound is classified as a probable human carcinogen (weight-of-evidence classification B2) based on sufficient data from animal bioassays (EPA 1990c). Benzo(b)fluoranthene produced tumors in mice after lung implantation (Deutsch-Wenzel et al. 1983), and intraperitoneal (LaVoie et al. 1987) or subcutaneous injection (Lacassagne et al. 1963). Although no human data are available that specifically link exposure to benzo(b)fluoranthene to human cancer, it is a component of mixtures that have been associated with human cancer (e.g., coal tar, soot, coke oven emissions, and cigarette smoke) (EPA 1984, 1990d).

Benzo(k)fluoranthene. The oral and inhalation slope factors for benzo(k)fluoranthene are 0.73 and 0.61 $[mg/(kg \cdot d)]^{-1}$, respectively. It is classified as a probable human carcinogen (weight-of-evidence classification B2) based on sufficient data from animal bioassays (EPA 1990e). Benzo(k)fluoranthene produced tumors after lung implantation in mice (Deutsch-Wenzel et al. 1983) and when administered with a promoting agent in skin-painting studies (Van Duuren et al. 1966). Although no human data are available that specifically link exposure to benzo(k)fluoranthene to human cancer, it is a component of mixtures that have been associated with human cancer (e.g., coal tar, soot, coke oven emissions, and cigarette smoke) (EPA 1984, 1990d).

Benzo(a)pyrene. An oral slope factor of 7.3 and an inhalation slope factor of 6.1 $[mg/(kg \cdot d)]^{-1}$ were estimated for benzo(a)pyrene based on multiple animal studies in rodent and nonrodent species demonstrating benzo(a)pyrene to be carcinogenic following administration by oral (Neal and Rigdon 1967), intratracheal (Feron et al. 1973; Kobayashi 1975), inhalation (Thyssen et al. 1981), and dermal (IARC 1973) routes. Benzo(a)pyrene has produced positive results in several *in vitro* bacterial and mammalian genetic toxicology assays (IARC 1973, 1983; Santodanato et al. 1981).

Human carcinogenicity data are inadequate for benzo(a)pyrene. Lung cancer has been shown to be induced by humans by various mixtures of PAHs known to contain benzo(a)pyrene, including cigarette smoke, roofing tar, and coke oven emissions. However, it is not possible to conclude from this information that benzo(a)pyrene is the responsible agent (EPA 1990d). Benzo(a)pyrene is classified as a probable human carcinogen (weight-of-evidence classification B2) (EPA 1990f).

Chrysene. The oral and inhalation slope factors used for chrysene are 0.073 and 0.061 $[mg/(kg \cdot d)]^{-1}$, respectively, based on the TEF approach. This compound is classified as a probable human carcinogen (weight-of-evidence classification B2) based on sufficient data from animal bioassays (EPA 1990g). Chrysene produced carcinomas and malignant lymphoma in mice after intraperitoneal injection (Wislocki et al. 1986; Buening et al. 1979) and skin carcinomas in mice following dermal exposure (Wynder and Hoffman 1959). Chrysene produced chromosomal abnormalities in hamsters and mouse germ cells after gavage exposure (Basler et al. 1977; Roszinsky-Kocher et al. 1979), positive responses in bacterial gene mutation assays (McCann et al. 1975; Tokiwa et al. 1977; Wood et al. 1977; LaVoie et al. 1979; Dunkel and Simmon 1980; Sakai et al. 1985; Kaden et al. 1979), and transformed mammalian cells exposed in culture (Marquardt and Heidelberger 1972; Pienta et al. 1977).

Although no human data are available that specifically link exposure to chrysene to human cancer, it is a component of mixtures that have been associated with human cancer (e.g., coal tar, soot, coke oven emissions, and cigarette smoke) (EPA 1984, 1990d; IARC 1983, 1984).

Dibenzo(a,h)anthracene. Using EPA's TEFs, the oral and inhalation slope factors used for dibenzo(a,h)anthracene are 7.3 and 6.1 $[mg/(kg \cdot d)]^{-1}$, respectively. It is classified as a probable human carcinogen (weight-of-evidence classification B2) based on sufficient data from animal bioassays (EPA 1990h). Dibenzo(a,h)anthracene produced carcinomas in mice following oral (Snell and Stewart 1962, 1963; Biancifiori and Caschera 1962; Berenblum and Haran 1955) or dermal exposure (Wynder and Hoffman 1959; Van Duuren et al. 1967), and injection site tumors in several species following subcutaneous (Bryan and Shimkin 1943; Lubet et al. 1981) or

intramuscular (EPA 1990d) administration. Dibenzo(a,h)anthracene has induced DNA damage and gene mutations in bacteria as well as gene mutations and transformation in several types of mammalian cell cultures (EPA 1990d).

Although no human data are available that specifically link exposure to dibenzo(a,h)anthracene to human cancer, it is a component of mixtures that have been associated with human cancer (e.g., coal tar, soot, coke oven emissions, and cigarette smoke) (EPA 1984, 1990d; IARC 1984).

Indeno(1,2,3-cd)pyrene. Using the Region IV TEFs, the oral and inhalation slope factors estimated for indeno(1,2,3-cd)pyrene are 0.73 and 0.61 $[mg/(kg \cdot d)]^{-1}$, respectively. This compound is classified as a probable human carcinogen (weight-of-evidence classification B2) based on sufficient data from animal bioassays (EPA 1990i). Indeno(1,2,3-cd)pyrene produced tumors in mice following lung implants (Deutsch-Wenzel et al. 1983), subcutaneous injection (Lacassagne et al. 1963), and skin tumors in dermal application studies (Hoffman and Wynder 1966; Rice et al. 1985a, 1986). Indeno(1,2,3-cd)pyrene tested positive in bacterial gene mutation assays (LaVoie et al. 1979; Hermann et al. 1980; Rice et al. 1985b).

Although no human data are available that specifically link exposure to indeno(1,2,3cd)pyrene to human cancer, it is a component of mixtures that have been associated with human cancer (e.g., coal tar, soot, coke oven emissions, and cigarette smoke) (EPA 1984, 1990d; IARC 1984).

Pyrene. Pyrene is not classifiable as to human carcinogenicity (weight-of-evidence classification D) based on no human data and inadequate data from animal bioassays (EPA 1991).

Uncertainty in Toxicity Measures

Noncarcinogenic. A chronic oral RfD of 0.03 $mg/(kg \cdot d)$ was estimated for pyrene based on kidney effects (renal tubular pathology and decreased kidney weights) in a mouse subchronic oral bioassay (EPA 1989g). A subchronic oral RfD of 0.3 $mg/(kg \cdot d)$ is listed in HEAST (EPA 1992e).

An uncertainty factor of 3000 was used in the estimation, which reflects 10 each for intra- and interspecies variability, 10 for the use of a subchronic study for chronic RfD derivation, and an additional 3 to account for the lack of both toxicity studies in a second species and developmental/reproductive study. The uncertainty factor for the subchronic value is 300.

The confidence in the principal study is medium, as it was a well-designed experiment that examined a variety of toxicological endpoints and identified both a NOAEL and LOAEL for the critical effect. Confidence in the data base is low, due to the lack of supporting subchronic, chronic, and developmental/reproductive studies. Accordingly, the confidence in the RfD is low (EPA 1993).

Carcinogenic. Several PAHs are strongly implicated in the proven association between smoking and lung cancer, between occupational exposure to coke-oven emissions, coal-tar pitch, mineral oils, and similar products and cancer of skin, lung, bladder, and the gastro-intestinal tract. However, in humans, because of the fact that exposure involves complex mixtures where there is interaction between a number of initiators and promoters, it is more difficult to assess the activity of individual members of the group (EPA 1980).

A number of PAHs have been found to induce tumors in experimental animals upon exposure by different routes of administration [e.g., B(a)A, B(b)F, B(k)F, B(a)P, DBA, and IP]. The more active congeners, like dibenzo(a,h)anthracene, induce tumors in multiple sites, and also act as transplacental carcinogens. For benzo(a)pyrene, lung and liver tumors have been induced by exposure to pregnant mice (Dean et al. 1986). Limited evidence is available to suggest that chrysene is carcinogenic in animals and the available data for pyrene provide no evidence that it is carcinogenic.

The skin of many rodent species, like the mouse, is extremely sensitive to chemical carcinogens of this type, and experience from epidemiological investigations tend to demonstrate a much lower sensitivity of human skin than that of the mouse, a finding that has been corroborated by investigations in monkeys (Pucknat 1981).

5.3.1.16 Polychlorinated biphenyls

Toxicity Profile

CAS Numbers: PCBs: 1336-36-3
PCB-1254: 11097-69-1
PCB-1260: 11096-82-5

PHYSICOCHEMICAL CHARACTERISTICS							
A. Wt.	MP (°C)	BP (°C)	SG (@25°C)	Water Sol (mg/L)	Log K_{oc}	Log K_{ow}	Vapor Pressure
1254: 327 1260:324-460	1254: 10 1260: 31	1254: 365-390 1260: 385-420	1254: 1.505 1260: 1.566	1254: 0.057 1260: 0.080	1254: 5.61 1260: 6.42	1254: 6.47 1260: 6.91	1254: 7.71E-05 1260: 4.05E-05

NONCARCINOGENIC TOXICITY VALUES							
Oral RfD (mg/kg/d)		Uncertainty Factor		Inhal. RfC (mg/m^3)		Uncertainty Factor	
Subchron	Chronic	Subchron	Chronic	Subchron	Chronic	Subchron	Chronic
NA	NA	NA	NA	NA	NA	NA	NA

CARCINOGENIC TOXICITY VALUES					
Cancer Slope Factor (CSF) $(mg/kg/d)^{-1}$		Unit Risk Factor (URF)		Weight-of-Evidence	Reference of study (RfD/RfC/ CSF/URF)
Oral	Inhalation	Oral $(\mu g/L)^{-1}$	Inhalation $(\mu g/m^3)^{-1}$		
PCBs: 7.70E+00	NA	PCBs: 2.20E-04	NA	B2	NA; NA; Norback and Weltman 1985

PCBs are a group of synthetic halogenated aromatic hydrocarbons. These organic compounds are produced by chlorination of a biphenyl with anhydrous chlorine in the presence of iron filings or ferric chloride as the catalysts. Aroclor 1254 and Aroclor 1260 are two of many Aroclor PCB formulations. Aroclor 1254 is a light yellow, viscous liquid with a weak odor, while Aroclor 1260 is a light yellow, sticky, soft resin with a weak odor (Eisler 1986).

General Toxicity. The toxicological properties of individual PCBs are influenced primarily by two factors: the partition coefficient based on solubility in N-octanol/water (K_{ow}); and steric factors, resulting from different patterns of chlorine substitution. In general, PCB isomers with high K_{ow} values and high numbers of substituted chlorines in adjacent positions constitute the greatest health concern. Biological responses to individual isomers or mixtures vary widely, even among closely related taxonomic species. The issue is further confounded by the presence of toxic impurities, such as polychlorinated dibenzofurans, which may have been formed during the PCB manufacturing process, or result from product usage (Eisler 1986).

No data are currently available to evaluate the chronic health hazard assessment for noncarcinogenic effects (RfDs or RfCs) (EPA 1989).

Evidence of Carcinogenicity. PCBs are probable human carcinogens (weight-of-evidence classification B2) based on hepatocellular carcinomas in three strains of rats and two strains of mice, and inadequate yet suggestive evidence of risk of liver cancer in humans by ingestion and inhalation or dermal contact (EPA 1989).

Although many studies of human carcinogenicity have been conducted, the data are inadequate due to confounding exposures or lack of exposure quantification (Bahn et al. 1976, 1977; NIOSH 1977; Brown and Jones 1981; Brown 1987; Bertazzi et al. 1987; Amano et al. 1984). Sufficient animal carcinogenicity data are available on PCB mixtures. Norback and Weltman (1985) fed 70 male and 70 female Sprague-Dawley rats a diet containing Aroclor 1260 (in corn oil) at 100 ppm for 16 months, followed by a 50-ppm diet for an additional 8 months. A sequential progression of liver lesions to hepatocellular carcinomas was observed.

EPA reported that although PCB congeners vary greatly as to their potency in producing biological effects, for purposes of estimating the carcinogenicity assessment, Aroclor 1260 is intended to be representative of all PCB mixtures. The Norback and Weltman (1985) study used an adequate number of animals, which were observed for their normal lifespan. Only one nonzero test dose was used. A risk estimate was calculated based on the numbers of malignant tumors alone, as called for in EPA's guidelines for carcinogen risk assessment. The slope factor thus derived is 7.7 mg/(kg • d), which is 26% less than that derived using combined malignant tumors and neoplastic nodules. The risk estimate is based on data obtained from Kimbrough et al. (1975). EPA indicated that PCB mixtures in drinking water may not be the same as mixtures introduced or used for testing carcinogenicity in animals (EPA 1989).

Uncertainty in Toxicity Measures

Noncarcinogenic. No data are available at this time to evaluate the health hazard assessment for noncarcinogenic effects (RfDs or RfCs) (EPA 1993).

Carcinogenic. PCBs are probable human carcinogens (weight-of-evidence classification B2) based on hepatocellular carcinomas in three strains of rats and two strains of mice and inadequate yet suggestive evidence of risk of liver cancer in humans by ingestion and inhalation or dermal contact (EPA 1993).

Although many studies of human carcinogenicity exist, the data are inadequate due to confounding exposures or lack of exposure quantification. Sufficient animal carcinogenicity data are available on PCB mixtures. Norback and Weltman (1985) fed 70 male and 70 female Sprague-Dawley rats a diet containing Aroclor 1260 (in corn oil) at 100 ppm for 16 months, followed by a 50-ppm diet for an additional 8 months. A sequential progression of liver lesions to hepatocellular carcinomas was observed.

EPA reported that although it is known that PCB congeners vary greatly as to their potency in producing biological effects, for purposes of estimating the carcinogenicity assessment, Aroclor 1260 is intended to be representative of all PCB mixtures. The Norback and Weltman (1985) study used an adequate number of animals, which were observed for their normal lifespan. Only one nonzero test dose was used. A risk estimate was calculated based on the numbers of malignant tumors alone, as called for in EPA's guidelines for carcinogen risk assessment. The slope factor thus derived is 5.7 mg/(kg • d), which is 26% less than that derived using combined malignant tumors and neoplastic nodules. The risk estimate is supported by an estimate based on data from Kimbrough et al. (1975). EPA pointed out that PCB mixtures in drinking water may not be the same as mixtures introduced or used for testing of carcinogenicity in animals (EPA 1993).

5.3.1.17 Uranium

Introduction

There are three naturally occurring isotopes of uranium: U-238, U-235, and U-234. Uranium is a solid silvery metal that forms complexes with oxygen and fluoride to form various solids ranging in color from brown-black (UO_2) to yellow-red (UO_3) and green (U_3O_8 and UF_4). Soluble compounds of uranium include uranium hexafluoride, uranyl fluoride, uranyl acetate, and uranyl nitrate. By weight, it is generally assumed that >99% of the total naturally occurring uranium is of the isotope U-238. By activity, as measured in picocuries (pCi), roughly 47.5% of total naturally occurring uranium is U-238. Another 47.5% is U-234, and the remaining 5% activity is that of U-235.

The average concentration of total uranium in soils is approximately 1.2 pCi/g, although this concentration differs with the bedrock from which the soil is formed (NCRP 1984).

Physicochemical Characteristics

Group: rare earth
Crystal structure: orthorhombic
Atomic number: 92
Atomic weight: 238.029
Shells: 2, 8, 18, 32, 21, 9, 2
Filling orbital: 5f3
Melt: 1132 C
Boil: 3818 C
Electronegativity: 1.38
Covalent radius: 1.42 A
Atomic volume: 12.59 cm^3/mol
First ionization potential: 6.05 V
Oxidation states: (6), 5, 4, 3
Density @ 293 K: 18.9 g/cm^3
Specific heat: 0.12 J/gK
Heat of vaporization: 477.0 kJ/mol
Heat of fusion: 8.520 kJ/mol
Electrical conductivity: 0.0380 10^6/cm ohm
Thermal conductivity: 0.276 W/cmK

Element Nuclides

Nuclide	Abundance	Weight	Half-Life
U-230	0%	230	20.8 d
U-231	0%	231	4.20 d
U-232	0%	232.0371	70 y
U-233	0%	233.0396	1.59E+05 y
U-234	5.5E-03%	234.0409	2.47E+05 y
U-235	0.72%	235.0439	7.04E+08 y
U-236	0%	236.0456	2.34E+07 y
U-237	0%	237	6.75 d
U-238	99.27%	238.0508	4.47E+09 y
U-239	0%	239	23.5 m
U-240	0%	240	14.1 h

Metabolic Data

Data from Reference Man (ICRP 1975)	
Uranium content of the body	90 μg
of the skeleton	59 μg
of the kidneys	7 μg
Daily intake in food and fluids	1.9 μg

Studies have been conducted on the uptake of uranium to the blood from the gastrointestinal tract. These studies show that the uptake for water-soluble inorganic compounds of uranium is much greater than for relatively insoluble compounds. Similarly, more soluble compounds are rapidly absorbed from the lung. Once in the blood, uranium is retained by the bone and kidneys. Uranium isotopes with shorter half-lives are assumed to be distributed uniformly over the bone surfaces; whereas, isotopes with longer half-lives are assumed to be uniformly distributed throughout the volume of mineral bone following their deposition in the skeleton (ICRP 1979).

Carcinogenic Toxicity

Uranium is listed as a Class A carcinogen, primarily based on the results of follow-up studies of populations exposed to the atomic bomb dropped on Hiroshima in 1945. Slope factors have been published in HEAST for assessing risk from exposure to U-238, U-235, and U-234. U-238 and U-235 are the parent isotopes for separate decay chains. The most probable effect of exposure to uranium would be an increase in bone sarcomas (BEIR IV).

Risk from exposure to the decay products of these isotopes can be addressed in part by using the "plus daughter" slope factors for U-238 and U-235, as indicated in HEAST. Slope factors are given for external exposure (primarily gamma radiation), oral exposure, and inhalation exposures (alpha and gamma radiation). Other components of risk from uranium exposure include the risk from the *progeny* of uranium, specifically radium-226 and radon-222.

Cancer Risk Slope Factors (HEAST 1992)	Oral risk/pCi	External risk/yr/pCi	Inhalation risk/pCi
U-234	1.6E-11	3.0E-11	2.6E-8
U-235	1.6E-11	2.4E-7	2.5E-8
U-235+D	1.6E-11	2.4E-07	2.5E-8
U-238	1.6E-11	2.1E-11	2.4E-8
U-238+D	2.8E-11	3.6E-08	5.2E-8

Decay Chains

U-234

U-234 → Th-230 → Ra-226 → Rn-222 → Po-218

Result of decaying 1 curie of U-234 for 365.25 days:

Nuclide	Half-life	Branching from parent	Fraction	Curies
U-234	24.45E+04 y	(parent)	1.0000E+00	1.0000E+00
Th-230	7.70E+04 y	1.00	9.0019E-06	9.0019E-06
Ra-226	1.60E+03 y	1.00	1.9496E-09	1.9496E-09
Rn-222	3.82E+00 d	1.00	1.8916E-09	1.8916E-09
Po-218	3.05E+00 m	1.00	1.8916E-09	1.8916E-09

U-235

U-235 → Th-231 → Pa-231 → Ac-227 → Fr-223
Ac-227 → Th-227

Result of decaying 1 curie of U-235 for 365.25 days:

Nuclide	Half-life	Branching from parent	Fraction	Curies
U-235	70.380E+07 y	(parent)	1.0000E+00	1.0000E+00
Th-231	2.552E+01 h	1.00	1.0000E+00	1.0000E+00
Pa-231	3.276E+04 y	1.00	2.1069E-05	2.1069E-05
Ac-227	2.177E+01 y	1.00	3.3047E-07	3.3047E-07
Fr-223	2.180E+01 m	0.0138	4.5599E-09	4.5599E-09
Th-227	1.872E+01 d	0.9862	2.8130E-07	2.8130E-07

U-238

U-238 → Th-234 → Pa-234m → Pa-234 → U-234
Pa-234m → U-234 → Th-230

Result of decaying 1 curie of U-238 for 365.25 days:

Nuclide	Half-life	Branching from parent	Fraction	Curies
U-238	44.680E+08 y	(parent)	1.0000E+00	1.0000E+00
Th-234	2.410E+01 d	1.000	9.9997E-01	9.9997E-01
Pa-234m	1.170E+00 m	1.000	9.9997E-01	9.9997E-01
Pa-234	6.700E+00 h	0.0016	1.6000E-03	1.6000E-03
U-234	2.445E+05 y	0.9984	2.5610E-06	2.5610E-06
U-234	2.445E+05 y	1.000	4.0991E-09	4.0991E-09
Th-230	7.700E+04 y	1.000	1.0545E-11	1.0545E-11

5.3.1.18 Cesium

Introduction

Cesium is a soft, light, silvery-white alkali metal. It was discovered in Germany in 1860. It is found in pollucite and as trace in one epidolite. It is used to remove air traces in vacuum tubes, and, since it readily ionizes, it is used as an ion rocket motor propellant.

Physicochemical Characteristics

Group: Alkali metals
Crystal structure: cubic body centered
Atomic number: 55
Atomic weight: 132.9054
Shells: 2, 8, 18, 18, 8, 1
Filling Orbital: 6s1
Melt: 28.5 C
Boil: 678.4 C
Electronegativity: 0.79
Covalent radius: 2.35 A
Atomic radius: 3.34 A
Atomic volume: 71.07 cm^3/mol
First ionization potential: 3.894 V
2nd ionization potential: 25.10 V
Oxidation states: 1
Density @ 293 K: 1.873 g/cm^3

Specific heat: 0.24 J/gK
Heat of vaporization: 67.740 kJ/mol
Heat of fusion: 2.092 kJ/mol
Electrical conductivity: 0.0489 10^6/cm ohm
Thermal conductivity: 0.359 W/cmK

Element Nuclides

Nuclide	Abundance	Weight	Half-Life
Cs-126	0%	126	1.64 m
Cs-129	0%	129	1.336 d
Cs-131	0%	131	9.69 d
Cs-132	0%	132	6.48 d
Cs-133	100%	33.9054	
Cs-134	0%	134	2.065 y
Cs-134m	0%	134	2.91 h
Cs-135	0%	135	2.3E+06 y
Cs-136	0%	136	13.16 d
Cs-137	0%	137	30.17 y
Cs-138	0%	138	32.2 m
Cs-139	0%	139	9.3 m

Metabolic Data

Data from Reference Man (ICRP 1975)

Cesium content of the body	1.5 mg
of the muscle	0.57 mg
of the bone	0.16 mg
Daily intake in food and fluids	10 μg

Studies conducted on cesium uptake to blood have indicated that commonly occurring compounds of cesium are rapidly and almost completely absorbed from the gastrointestinal tract. In addition, available evidence indicates that cesium is distributed uniformly throughout all organs and tissues in the body (ICRP 1979).

Carcinogenic Toxicity

Cesium is listed as a Class A carcinogen. Slope factors have been published in HEAST for assessing risk from exposure to several cesium isotopes. Cs-137 is the parent isotope for a separate decay chain.

Risk from exposure to the decay products can be addressed in part by using the "plus daughter" slope factors for Cs-137, as indicated in HEAST. Slope factors are given for external exposure (primarily gamma radiation), oral exposure, and inhalation exposures (alpha and gamma radiation). Other components of risk from exposure include the risk from the *progeny* of cesium.

Cancer Risk Slope Factors (HEAST 1992)	Oral risk/pCi	External risk/pCi	Inhalation risk/pCi
Cs-137	2.8E-11	0.0E+00	1.9E-11
Cs-137+D	2.8E-11	2.0E-06	1.9E-11

Decay Chains

Cs-137

Cs-137 ——————▸ Ba-137m ——————▸ stable

Result of decaying 1 curie of Cs-137 for 0.0 days:
No daughters activity because parent fraction is >0.9999999

Nuclide	Half-life	Branching from parent	Fraction	Curies
Cs-137	30.17E+00 y	(parent)	1.0000E+00	1.0000E+00
Ba-137m	2.552E+00 m	0.9460	0.0	0.0

5.3.2 Derivation of Alternate RfDs for Mercury Compounds

Mercury, a toxic heavy metal, is the principal contaminant of concern at EFPC. No EPA-verified oral RfDs or inhalation RfCs are currently available (EPA 1993). The Agency RfD work group is currently evaluating the chronic health effects data on mercury for its potential noncarcinogenic effects. In the interim, however, EPA has published a tentative oral and inhalation RfD of 0.0003 mg/(kg•d) for inorganic mercury based on the induction of immune-type glomerulonephritis (GN) by mercuric chloride in Brown-Norway rats (EPA 1991f). A

critical problem with this interim RfD is the selection of an autoimmune-type disease observed in sensitive rat strains exposed to very low levels of mercury as the basis for its derivation.

As a principal contaminant of concern, information on the physicochemical properties of mercury was considered crucial in order to more accurately estimate the public health and environmental hazards posed by mercury contamination. During the RI, chemical analysis of the soil samples collected from the floodplains indicated that mercury was predominantly present in an insoluble form, probably as mercuric sulfide (Revis et al. 1989). Since the toxicity of mercury closely parallels its solubility in the aqueous media, and the existing RfD for mercury is based on the toxicities of mercuric chloride (a highly soluble form of mercury), a detailed examination of the toxicity of these two inorganic mercurial species was considered necessary.

The EFPC risk assessment team worked to identify (derive) plausible alternative RfDs for inorganic mercury. Instead of a single RfD for all of the inorganic species, an effort was made to derive an alternative oral RfD for mercuric chloride as a typical soluble inorganic species and for mercuric sulfide as the typical insoluble form of mercury. It is believed that the insoluble mercuric sulfide species predominates in EFPC.

As a result, alternate RfDs of 1×10^{-3} for mercuric chloride and 4×10^{-2} for mercuric sulfide were derived and are presented for review in Appendix N. However, as a result of discussions with EPA Region IV, it has been agreed that no alternate RfDs will be used in the BRA risk calculations unless the EPA Office of Research and Development has given its approval. The RfD of 3×10^{-4}, currently presented in the EPA HEAST compilation (EPA 1991f)*, has been used in the risk calculations. Nevertheless, these alternate RfDs are important and show that, depending on the form of mercury and the studies chosen, the appropriate toxicity value for mercury at EFPC might decrease as much as two orders of magnitude. This is an important uncertainty in the risk calculations for mercury.

5.3.3 Chemicals for Which no EPA Toxicity Values are Available

PAHs. In the past, EPA's approach has been to assume that all PAHs identified in a mixture have carcinogenic potencies equal to that of benzo(a)pyrene. This approach has little scientific backing and may result in great overestimation of risk. In fact, numerous studies have shown that benzo(a)pyrene is one of the more potent PAH carcinogens, and is more potent than many mixtures of PAHs.

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*_Note:_ There is not a current listing on the IRIS data base.

The relative potency approach for carcinogens involves the use of TEFs, which assign specific PAHs an equivalence factor relative to that of benzo(a)pyrene. The latter is assigned a TEF of one. In addition, the relative potencies are assumed to be additive. This assumes that agents induce cancer by the same or a similar mechanism. This allows for the estimation of cancer risk to combined mixtures simply by adding the doses.

This approach has been documented in several literature sources, one of which is a report prepared for EPA by ICF-Clement Associates (1988). The approach also has been adopted by EPA Region IV as their standard protocol for evaluating carcinogenicity in PAHs (EPA 1992a).

In the studies conducted, mice were evaluated for tumor response to benzo(a)pyrene and PAH mixtures. Relative potency estimates can be determined from experiments in which benzo(a)pyrene and other PAHs are concurrently tested. It is then possible to estimate ratios of exposure-induced mutation rates per unit of exposure, and use dose additivity to estimate exposure to multiple PAH compounds.

In the EFPC BRA, as specified in the EPA Region IV Guidance (EPA 1992a), the following TEFs have been used to convert the oral and inhalation slope factors of each PAH to an equivalent benzo(a)pyrene slope factor:

Compound	TEF
benzo(a)pyrene	1.0
benzo(a)anthracene	0.1
benzo(b)fluoranthene	0.1
benzo(k)fluoranthene	0.1
chrysene	0.01
dibenzo(a,h)anthracene	1.0
indeno(1,2,3-cd)pyrene	0.1

In addition, for noncarcinogenic effects, the above PAHs were assumed to have oral RfDs (both chronic and subchronic) equal to that of pyrene. This convention was recommended by EPA Region IV.

Lead. No toxicity values are currently available from EPA for lead exposures. Considerable uncertainty surrounds the designation of a single point estimate for acceptable or unacceptable health effects due to lead. EPA recommends that risk characterization for lead be based on an alternative approach in which blood lead uptake is compared to the latest Center for Disease Control guideline of 10 μg/dL blood lead in children. EPA has developed a computer

program, LEAD 0.60 (EPA 1991e) that estimates blood lead uptake from various environmental sources. Blood lead levels are expressed as probability percents, which is the probability that levels will be either less than or greater than the target blood lead level of 10 μg/dL. LEAD 0.60 analyzes blood lead uptake in children, the most sensitive receptors of lead exposures, and is not currently applicable to adults.

5.3.4 General Uncertainties Related to Toxicity Information

This section discusses general uncertainties related to the toxicity information. Although EPA provides toxicity values that are point estimates, a significant amount of uncertainty may surround these point estimates. Identification of the sources of this uncertainty enables the risk assessor to establish the degree of confidence associated with the toxicity measures. The following paragraphs discuss these sources.

Uncertainty is inherent within the toxicity assessment and is primarily due to differences in study design, species, sex, routes of exposure, or dose-response relationships. A major source of uncertainty involves using toxicity values based on experimental studies that substantially differ from typical human exposure scenarios. The derivation of the toxicity values must take into account such differences as: 1) using dose-response information from animal studies to predict effects in humans, 2) using dose-response information from high-dose studies to predict adverse health effects from low doses, 3) using data from short-term studies to predict chronic effects, and 4) extrapolating from specific to general populations.

The CSFs in particular are based on studies that may differ greatly from realistic situations. Experimental cancer bioassays typically expose animals to very high levels of chemicals (i.e., the maximum tolerated dose) for their entire lifetime. After the appropriate studies have been identified, the slope factor is calculated as the 95% UCL of the slope of the dose-response curve. This introduces conservatism into the risk assessment.

The derivation of RfDs generally involves the use of animal studies. Uncertainty factors ranging from 1 to 10,000 are used in the calculation of the RfD to provide an extra level of public health protection. The factors used reflect scientific judgment regarding the type of study from which the value has been derived (e.g., animal or human, chronic or acute). The scientific basis for this practice is somewhat uncertain. In general, high uncertainty factors are meant to bias the results conservatively so that the RfD will not result in adverse health effects.

Toxicological endpoints are constantly being identified and revised as appropriate. As data are developed, differences between species, sex, route of exposure, duration of exposure, study

design, and dose-response relationships are better understood and a higher level of confidence is gained. For the purposes of risk assessment, values based on human data generally have a higher degree of confidence than do those derived from animal data.

In the risk characterization process, risks are summed (separately for carcinogens and noncarcinogens) to estimate potential risks associated with the simultaneous exposure to multiple chemicals. In the case of carcinogens, this gives carcinogens with a Class B or Class C weight-of-evidence the same weight as carcinogens with a Class A weight-of-evidence. It also equally weights slope factors derived from animal data with those derived from human data. Uncertainties in the combined risks also are compounded because RfDs and CSFs do not have equal accuracy or levels of confidence and are not based on the same severity of effect.

Oral toxicity values have been used to evaluate dermal exposures, since dermal toxicity values are unavailable. In the absence of estimates of gastrointestinal absorption, this approach is recommended by EPA (1992b). This introduces uncertainty because risks associated with dermal contact effects may not be accurately estimated using oral toxicity data based on oral effects. Since research on this topic is limited, it is difficult to predict the bias of the uncertainty.

As explained at the beginning of this section, no adjustment to the toxicity measures has been made to express both dose and the toxicity measure in the same terms (i.e., absorbed or administered dose). This adds to the uncertainty associated with the toxicity measures. In theory, all exposure estimates and toxicity measures would be expressed as absorbed dose.

Toxicity values are not available for all of the carcinogenic PAHs. However, values have been assigned to several carcinogenic PAHs based upon TEFs, which relate the carcinogenicity of each PAH to the carcinogenicity of benzo(a)pyrene. This approach, although currently under review by EPA, is thought to be more realistic than the alternative method of assuming that all carcinogenic PAHs have a potency factor equal to that of benzo(a)pyrene.

5.3.5 Summary of Toxicity Information

The toxicity values used in the BRA are summarized in Table 5.19. All values in this table, except where indicated in the footnotes, are from IRIS or HEAST (see columns entitled "Source").

Table 5.19. Toxicity measures for waste site evaluation: ingestion and inhalation pathways

	NONCARCINOGENIC												
	Oral Route (mg/kg/day)		Uncertainty Factor Oral Route	Uncertainty Factor Oral Route		Inhalation Route (mg/m3)		Inhalation Route (mg/kg/day)		Uncertainty Factor Inhal. Route	Uncertainty Factor Inhal. Route		Noncarcinogenic
COMPOUND	RfD-S[a]	RfD-C[a]	(subchronic)	(chronic)	Source (Oral)	RfC-S[b]	RfC-C[b]	RfD-S[b]	RfD-C[b]	(subchronic)	(chronic)	Source (Inhal.)	Target Organ and Critical Effect
INORGANICS													
Arsenic	3.00E-04	3.00E-04	3	3	d,e	--	--	--	--	--	--	--	skin; keratosis, hyperpigmentation
Barium	7.00E-02	7.00E-02	3	3	d,e	5.00E-03	5.00E-04	1.43E-03	1.43E-04	100	1000	d	*oral*-cardiovasc. sys.; inc. blood pressure. *inhal.*-fetotoxic
Beryllium	5.00E-03	5.00E-03	100	100	d,e	--	--	--	--	--	--	--	no observed effects
Cadmium (food)	--	1.00E-03	--	10	e	--	--	--	--	--	--	--	kidney; proteinuria
Cadmium (water)	--	5.00E-04	--	10	e	--	--	--	--	--	--	--	kidney; proteinuria
Chromium (III)	1.00E+00	1.00E+00	1000	100	d,e	--	--	--	--	--	--	--	no observed effects
Chromium (VI)	2.00E-02	5.00E-03	100	500	d,e	--	--	--	--	--	--	--	no observed effects
Cobalt	--	--	--	--	--	--	--	--	--	--	--	--	--
Copper	3.70E-02	3.70E-02	--	--	d,j	--	--	--	--	--	--	--	Gastrointestinal system; irritation
Cyanide	2.00E-02	2.00E-02	500	100	d,e	--	--	--	--	--	--	--	thyroid, nerve; weight loss, myelin degen.
Lead	--	--	--	--	f	--	--	--	--	--	--	--	CNS, blood
Manganese (food)	1.00E-01	1.40E-01	1	1	d,e	4.00E-04	4.00E-04	1.14E-04	1.14E-04	900	300	d,e	*oral*-CNS effects. *inhal.*-respiratory effects
Manganese (water)	1.00E-01	5.00E-03	1	1	d,e	4.00E-04	4.00E-04	1.14E-04	1.14E-04	900	300	d,e	*oral*-CNS effects. *inhal.*-respiratory effects
Mercury	3.00E-04	3.00E-04	1000	1000	d	3.00E-04	3.00E-04	8.57E-05	8.57E-05	30	30	d	*oral*-CNS; neurotoxicity. *inhal.*-kidney effects
Nickel	2.00E-02	2.00E-02	300	300	d,e,o	--	--	--	--	--	--	--	dec. body and organ weight
Silver	5.00E-03	5.00E-03	3	3	d,e	--	--	--	--	--	--	--	skin; argyria
Vanadium	7.00E-03	7.00E-03	100	100	d	--	--	--	--	--	--	--	no observed effects
Zinc	3.00E-01	3.00E-01	3	3	d,e,o	--	--	--	--	--	--	--	blood; anemia
ORGANICS													
Acenaphthene	6.00E-01	6.00E-02	300	3000	d,e	--	--	--	--	--	--	--	liver; hepatotoxicity
Acenaphthylene	3.00E-01	3.00E-02	--	--	h	--	--	--	--	--	--	--	--
Acetone	1.00E+00	1.00E-01	100	1000	d,e	--	--	--	--	--	--	--	inc. liver, kidney weights, nephrotoxicity
Aldrin	3.00E-05	3.00E-05	1000	1000	d,e	--	--	--	--	--	--	--	liver; lesions, toxicity
Anthracene	3.00E+00	3.00E-01	300	3000	d,e	--	--	--	--	--	--	--	no observed effects
Aroclor-1254	--	--	--	--	--	--	--	--	--	--	--	--	--
Aroclor-1260	--	--	--	--	--	--	--	--	--	--	--	--	--
Benzo(a)anthracene	3.00E-01	3.00E-02	--	--	h	--	--	--	--	--	--	--	--
Benzo(a)pyrene	3.00E-01	3.00E-02	--	--	h	--	--	--	--	--	--	--	--
Benzo(b)fluoranthene	3.00E-01	3.00E-02	--	--	h	--	--	--	--	--	--	--	--
Benzo(g,h,i)perylene	3.00E-01	3.00E-02	--	--	h	--	--	--	--	--	--	--	--

Table 5.19 (continued)

	NONCARCINOGENIC													
	Effects Oral Route (mg/kg/day)		Uncertainty Factor Oral Route	Uncertainty Factor Oral Route		Effects Inhalation Route (mg/m3)		Effects Inhalation Route (mg/kg/day)		Uncertainty Factor Inhal Route	Uncertainty Factor Inhal Route		Noncarcinogenic
COMPOUND	RfD-S[a]	RfD-C[a]	(subchronic)	(chronic)	Source (Oral)	RfC-S[b]	RfC-C[b]	RfC-S[b]	RfC-C[b]	(subchronic)	(chronic)	Source (Inhal.)	Target Organ and Critical Effect
ORGANICS (Cont.)													
Benzo(k)fluoranthene	3.00E-01	3.00E-02	--	--	h	--	--	--	--	--	--	--	--
Benzoic Acid	4.00E+00	4.00E+00	1	1	d,c	--	--	--	--	--	--	--	no observed effects
Bis(2-ethylhexyl)phthalate	2.00E-02	2.00E-02	1000	1000	d,c	--	--	--	--	--	--	--	liver; weight inc.
Butyl benzyl phthalate	2.00E+00	2.00E-01	100	1000	d,c	--	--	--	--	--	--	--	liver; weight changes
Chloroform	1.00E-02	1.00E-02	1000	1000	d,c	--	--	--	--	--	--	--	liver; lesions, fatty cyst formation
Chrysene	3.00E-01	3.00E-02	--	--	h	--	--	--	--	--	--	--	--
4,4-DDD	--	--	--	--	--	--	--	--	--	--	--	--	--
4,4-DDT	5.00E-04	5.00E-04	100	100	d,c	--	--	--	--	--	--	--	liver; lesions
Dibenzo(a,h)anthracene	3.00E-01	3.00E-02	--	--	h	--	--	--	--	--	--	--	--
Dibenzofuran	--	--	--	--	--	--	--	--	--	--	--	--	--
Di-n-butyl phthalate	1.00E+00	1.00E-01	100	1000	d,c	--	--	--	--	--	--	--	inc. mortality
Dieldrin	5.00E-05	5.00E-05	100	100	d,c	--	--	--	--	--	--	--	liver; lesions
Diethyl phthalate	8.00E+00	8.00E-01	100	1000	d,c	--	--	--	--	--	--	--	dec body growth, dec. organ weight
Endosulfan	2.00E-04	--	1000	--	d,q	--	--	--	--	--	--	--	kidney; toxicity, lesions
Endosulfan Sulfate	--	--	--	--	--	--	--	--	--	--	--	--	--
Endrin	3.00E-04	3.00E-04	100	100	d,c	--	--	--	--	--	--	--	CNS, liver; convulsions, lesions
Fluoranthene	4.00E-01	4.00E-02	300	3000	d,c	--	--	--	--	--	--	--	kidney, liver, blood; inc. weight, hematological changes
Fluorene	4.00E-01	4.00E-02	300	3000	d,c	--	--	--	--	--	--	--	erythrocytes; dec. counts
gamma-BHC	3.00E-03	3.00E-04	100	1000	d,c	--	--	--	--	--	--	--	liver, kidney; toxicity
Heptachlor Epoxide	1.30E-05	1.30E-05	1000	1000	d,c	--	--	--	--	--	--	--	liver; inc. relative weight
Indeno(1,2,3-cd)pyrene	3.00E-01	3.00E-02	--	--	h	--	--	--	--	--	--	--	--
Methylene Chloride	6.00E-02	6.00E-02	100	100	d,c	3.00E+00	3.00E+00	8.57E-01	8.57E-01	100	100	d	*oral and inhal.*-liver; toxicity
2-Methylnaphthalene	3.00E-01	3.00E-02	300	--	h	--	--	--	--	--	--	--	skin effects
Naphthalene	--	--	--	--	n	--	--	--	--	--	--	--	dec. body weight
PCB	--	--	--	--	--	--	--	--	--	--	--	--	--
Phenanthrene	3.00E-01	3.00E-02	--	--	h	--	--	--	--	--	--	--	--
Pyrene	3.00E-01	3.00E-02	300	3000	d,c	--	--	--	--	--	--	--	kidney; red. weight, renal tubular pathology
Tetrachloroethylene	1.00E-01	1.00E-02	100	1000	d,c	--	--	--	--	--	--	--	liver; hapatotoxicity
1,1,1-Trichloroethane	9.00E-01	9.00E-02	100	1000	d	1.00E+01	1.00E+00	2.86E+00	2.86E-01	100	1000	d	*oral and inhal.*-liver; hepatotoxicity
Trichloroethylene	--	--	--	--	--	--	--	--	--	--	--	--	--

Table 5.19 (continued)

	CARCINOGENIC							
COMPOUND	Cancer Slope Factor (CSF): Oral Route (mg/kg/day)-1	Weight-of-Evidence	Unit Risk Factor Oral Route (ug/l)-1	Source (Oral)	Cancer Slope Factor (CSF): Inhalation Route (mg/kg/day)-1	Unit Risk Factor Inhalation Route (ug/m3)-1	Weight-of-Evidence	Source (Inhal.)
INORGANICS								
Arsenic	1.75E+00	[A]	5.00E-05	c,e	5.00E+01	4.30E-03	[A]	d,c
Barium	--		--	--	--	--		--
Beryllium	4.30E+00	[B2]	1.20E-04	c	8.40E+00	2.40E-03	[B2]	d,c
Cadmium (food)	--		--	--	6.10E+00	1.80E-03	[B1]	d,c
Cadmium (water)	--		--	--	6.10E+00	1.80E-03	[B1]	d,c
Chromium (III)	--		--	--	--	--		--
Chromium (VI)	--		--	--	4.10E+01	1.20E-02	[A]	d,c
Cobalt	--		--	--	--	--		--
Copper	--	[D]	--	c	--	--		--
Cyanide	--	[D]	--	c	--	--		--
Lead	--	[B2]	--	c	--	--		--
Manganese (food)	--	[D]	--	c	--	--		--
Manganese (water)	--	[D]	--	c	--	--		--
Mercury	--	[D]	--	c	--	--		--
Nickel	--		--	--	--	--		--
Silver	--	[D]	--	c	--	--		--
Vanadium	--		--	--	--	--		--
Zinc	--	[D]	--	c	--	--		--
ORGANICS								
Acenaphthene	--		--	--	--	--		--
Acenaphthylene	--	[D]	--	c	--	--		--
Acetone	--	[D]	--	c	--	--		--
Aldrin	1.70E+01	[B2]	4.90E-04	c	1.70E+01	4.90E-03	[B2]	d,c
Anthracene	--	[D]	--	c	--	--		--
Aroclor-1254	7.70E+00	[B2]	2.20E-04	c,m	--	--		--
Aroclor-1260	7.70E+00	[B2]	2.20E-04	c,m	--	--		--
Benzo(a)anthracene	7.30E-01	[B2]	--	i	6.10E-01	--	[B2]	i
Benzo(a)pyrene	7.30E+00	[B2]	2.10E-04	c	6.10E+00	1.70E-03	[B2]	d
Benzo(b)fluoranthene	7.30E-01	[B2]	--	i	6.10E-01	--	[B2]	i
Benzo(g,h,i)perylene	--	[D]	--	c	--	--		--

Table 5.19 (continued)

CARCINOGENIC

COMPOUND	Cancer Slope Factor (CSF): Oral Route (mg/kg/day)-1	Weight-of-Evidence	Unit Risk Factor Oral Route (ug/l)-1	Source (Oral)	Cancer Slope Factor (CSF): Inhalation Route (mg/kg/day)-1	Unit Risk Factor Inhalation Route (ug/m3)-1	Weight-of-Evidence	Source (Inhal.)
ORGANICS (Cont.)								
Benzo(k)fluoranthene	7.30E-01	[B2]	--	i	6.10E-01	--	[B2]	i
Benzoic Acid	--	[D]	--	c	--	--		--
Bis(2-ethylhexyl)phthalate	1.40E-02	[B2]	4.00E-07	c	--	--		--
Butyl benzyl phthalate	--	[C]	--	c	--	--		--
Chloroform	6.10E-03	[B2]	1.70E-07	c	8.10E-02	2.30E-05	[B2]	d,c
Chrysene	7.30E-02	[B2]	--	i	6.10E-02	--	[B2]	i
4,4-DDD	2.40E-01	[B2]	6.90E-06	c	--	--		--
4,4-DDT	3.40E-01	[B2]	9.70E-06	c	3.40E-01	9.70E-05	[B2]	d,c
Dibenzo(a,h)anthracene	7.30E+00	[B2]	--	i	6.10E+00	--	[B2]	i
Dibenzofuran	--	[D]	--	c	--	--		--
Di-n-butyl phthalate	--	[D]	--	c	--	--		--
Dieldrin	1.60E+01	[B2]	4.60E-04	c	1.60E+01	4.60E-03	[B2]	d,c
Diethyl phthalate	--	[D]	--	c	--	--		--
Endosulfan	--		--	--	--	--		--
Endosulfan Sulfate	--		--	--	--	--		--
Endrin	--	[D]	--	c	--	--		--
Fluoranthene	--	[D]	--	c	--	--		--
Fluorene	--	[D]	--	c	--	--		--
gamma-BHC	1.30E+00	[C]	3.70E-05	d	--	--		--
Heptachlor Epoxide	9.10E+00	[B2]	2.60E-04	c	9.10E+00	2.60E-03	[B2]	d,c
Indeno(1,2,3-cd)pyrene	7.30E-01	[B2]	--	i	6.10E-01	--	[B2]	i
Methylene Chloride	7.50E-03	[B2]	2.10E-07	c	1.65E-03	4.70E-07	[B2]	g,c
2-Methylnaphthalene	--		--	--	--	--		--
Naphthalene	--	[D]	--	c	--	--		--
PCB	7.70E+00	[B2]	2.20E-04	c	--	--		--
Phenanthrene	--	[D]	--	c	--	--		--
Pyrene	--	[D]	--	c	--	--		--
Tetrachloroethylene	--		--	--	--	--		--
1,1,1-Trichloroethane	--	[D]	--	c	--	--		--
Trichloroethylene	--		--	--	--	--		--

Table 5.19 (continued)

EPA Slope Factors for Radionuclides[7]

COMPOUND	Slope Factor Ingestion Pathway (Risk/pCi)	Slope Factor Inhalation Pathway (Risk/pCi)	Slope Factor Ground Surface (Risk/yr per pCi/g)
RADIONUCLIDES			
Americium 241	2.40E−10	3.20E−08	4.90E−09
Cesium 137	2.80E−11	1.90E−11	0.00E+00
Cesium 137+D	2.80E−11	1.90E−11	2.00E−06
Cobalt 60	1.50E−11	1.50E−10	8.60E−06
Neptunium 237+D	2.20E−10	2.90E−08	4.30E−07
Uranium 234	1.60E−11	2.60E−08	3.00E−11
Uranium 235	1.60E−11	2.50E−08	2.40E−07
Uranium 235+D	1.60E−11	2.50E−08	2.40E−07
Uranium 238	1.60E−11	2.40E−08	2.10E−11
Uranium 238+D	2.80E−11	5.20E−08	3.60E−08

Table 5.19 (continued)

[a]RfD–S: Reference dose for subchronic exposure, oral route. RfD–C: Reference dose for chronic (long–term) exposure, oral route.

[b]RfC–S: Reference concentration for subchronic (short–term) exposure, inhalation route. RfC–C: Reference concentration for chronic (long–term) exposure, inhalation route. Inhalation RfCs have been converted to inhalation RfDs by multiplying by 20 m^3/day and dividing by 70 kg.

[c]EPA IRIS Data Base (as of March 1993).

[d]USEPA ORD Health Effects Assessment Summary Tables (HEAST) FY 1992 Annual (March 1992), Supplement No. 1 (July 1992), and Supplement No. 2 (November 1992).

[e]The oral unit risk has been proposed by EPA. The oral slope factor was calculated from the unit risk by assuming an ingestion of 2 liters of water per day by a 70 kg adult.

[f]EPA has not developed a reference dose for lead. EPA recommends use of the lead biokinetic model to estimate blood lead levels for the purposes of risk assessment.

[g]The inhalation slope factor for methylene chloride was calculated from the inhalation unit risk by assuming an inhalation rate of 20 m^3/day by a 70 kg adult.

[h]In the absence of toxicity data, the RfDs for pyrene have been adopted for this compound.

[i]The cancer slope factor for this compound has been estimated by multiplying the cancer slope factor for benzo(a)pyrene by a toxicity equivalence factor (USEPA Region IV, Interim Guidance, February 11, 1992). This method was also adopted for the inhalation slope factors (personal communication with Dr. Elmer Aiken, USEPA Region IV, October 19, 1992).

[j]EPA Office of Drinking Water MCL of 1.3 mg/L has been converted to intake estimate of 3.7E–02 mg/kg–day by assuming ingestion of 2 liters of water/day by a 70 kg adult.

[k]In the absence of data, the cancer slope factor for PCBs as a class of compounds has been adopted for Aroclor–1254 and Aroclor–1260.

[l]EPA has withdrawn the chronic and subchronic oral RfDs for naphthalene from HEAST (Nov. 1992).

[m]Values are for metals in the form of soluble salts.

[n]Radionuclide slope factors from HEAST January 1992.

[o]EPA has withdrawn the chronic oral RfD for endosulfan from IRIS (Dec. 1992).

RELATIVE POTENCY OF PAHs
(from USEPA Region IV, Interim Guidance, February 1992)

PAH	Relative Potency
Benzo(a)pyrene	1.000
Benzo(a)anthracene	0.100
Benzo(b)fluoranthene	0.100
Benzo(k)fluoranthene	0.100
Chrysene	0.010
Dibenzo(a,h)anthracene	1.000
Indeno(1,2,3–cd)pyrene	0.100

5.4 RISK CHARACTERIZATION

5.4.1 Overview

The last step in the human health risk assessment is risk characterization, which is the process of integrating the results of the exposure and toxicity assessments (i.e., comparing estimates of dose with appropriate toxicity measures to determine the likelihood of adverse effects in exposed populations). A hierarchical approach has been chosen for the baseline human health risk assessment of EFPC. The elements of this approach are described below, and are in keeping with both EPA and ORNL CRAC guidance.

5.4.1.1 Tier I: screening-level deterministic assessment using Phase Ia monitoring data

All chemicals and radionuclides observed in environmental media are included in the risk assessment. Data obtained from the Phase Ia study for three transects across EFPC are used in a preliminary screening-level risk assessment using both "most likely exposure" (MLE: average or 50%) and "reasonable maximum exposure" (RME: upper-bound or 95%) assumptions. MLE and RME point estimates are adopted for environmental concentrations as well as for all input variables used in the exposure assessment equations. Use of RME and average estimates follows the recent guidance from EPA (1992c) as well as recommendations from ORNL CRAC. ORNL suggests the use of both a conservative and nonconservative screening-level assessment to bound the risk estimates and identify exposure pathways of concern (i.e., the conservative and nonconservative estimates correspond to the RME and MLE evaluations, respectively). Risk estimates make use of EPA-verified toxicity measures (point estimates) and are based on estimates of chronic intake or dose. A supplementary assessment of subchronic risk to young children and adults also is conducted, complying with a request from ORNL CRAC.

5.4.1.2 Tier II: deterministic assessment using Phase Ia and Ib monitoring data

In the Tier II assessment, COCs are selected based on the results of the Tier I assessment and the use of an EPA concentration-toxicity screening analysis. The concentration-toxicity screen evaluated the full monitoring data set (i.e., Phase Ia and Ib). Risk assessment was conducted using both MLE and RME values. (Note, in compliance with a request from EPA Region IV, only risk estimates based upon RME point estimates are presented in the risk characterization section of this report). RME point estimates are adopted for environmental concentrations, as well as for all input variables used in the exposure assessment equations. All available monitoring data (i.e., Phases Ia and Ib) are used in deriving exposure point estimates. Risk estimates use EPA-verified toxicity measures (point estimates) and are based on estimates of chronic intake or dose.

The current method for evaluating lead exposures differs from that used for the other chemicals and radionuclide substances. For this reason, lead exposures at EFPC are explored in a separate portion of the Tier II risk characterization.

Human receptors included in the Tier II risk characterization include both adults and children, for each land use area, for both current and future exposure. Locations included in the Tier II risk assessment are each of the nine segments at EFPC, the SLB, and, as a reference area for comparison to background, Hinds Creek.

5.4.1.3 Tier III: probabilistic assessment and quantitative uncertainty analysis

The Tier III assessment is a probabilistic evaluation of risks to human health using Monte Carlo simulation. In addition to the RME and MLE estimates, the risk assessment of EFPC uses PDFs and Monte Carlo techniques to generate risk estimates in the form of probability distributions. PDFs for key input variables in the exposure and risk characterization equations are used instead of point estimates. Tier III risk estimates use EPA-verified toxicity measures (point estimates) and are based on estimates of chronic intake or dose.

As noted above, EPA Region IV requests RME estimates as the principal basis of this risk evaluation, and that the risk characterization section present only RME point estimates. Therefore, the results of the Monte Carlo analysis (i.e., the Tier III assessment) are presented in Sect. 5.5 (uncertainty analysis), and supplemental tables are provided in the appendixes.

Human receptors in the Tier III assessment include both adults and children in a residential setting, using exposure point concentration values derived from the statistical distribution of exposure point concentrations that were developed for Segment 4.

5.4.2 Risk Characterization Methods

The risk characterization for EFPC includes evaluation of both nonradioactive materials (chemical hazards) as well as radionuclides. The methods used to characterize risks associated with exposure to these two types of contaminants are presented in the following sections.

5.4.2.1 Risk characterization methods for chemical (nonradioactive) contaminants

EPA has previously established the procedure for calculating risk associated with exposure to carcinogenic chemicals (EPA 1986b,c; 1989a). A nonthreshold, dose-response model is used to calculate a CSF (which mathematically is the slope of the dose-response curve) for each

chemical. To derive an estimate of risk, the CSF is then multiplied by the estimated chronic daily dose experienced by the exposed individual:

$$Risk = CDI \times CSF \quad (33)$$

where

Risk = upper-bound estimate of the excess lifetime cancer risk to an individual (unitless probability),
CDI = chronic daily dose averaged over a 70-year period (mg/kg bw/d),
CSF = 95% upper-bound estimate of the slope of the dose-response curve $(mg/kg\ bw/d)^{-1}$.

The CSF is used to convert estimates of daily intake or dose averaged over a lifetime to incremental excess risk of an individual developing cancer. EPA notes that use of this equation assumes that the dose-response relationship is linear in the low-dose portion of the multistage model dose-response curve (EPA 1989a). Given this assumption, the CSF is constant and risk is directly proportional to intake.

EPA indicates that equation (34) is valid only at risk levels greater than 1×10^{-2}. The Agency recommends use of the following equation (based on the "one-hit" model of carcinogenesis (EPA 1989a) as an alternative at sites where exposure and intakes are projected to be quite high (i.e., in cases where risk levels may exceed 1×10^{-2}):

$$Risk = 1 - \exp(-CDI \times CSF) \quad (34)$$

In evaluating risk of exposure to more than one carcinogen, the risk measure for each compound is summed (in the absence of information on antagonistic or synergistic effects) to provide an overall estimate of total carcinogenic risk (EPA 1989a):

$$Risk_T = \sum_{i=1}^{n} Risk_i \quad (35)$$

where

$Risk_T$ = the combined excess lifetime cancer risk across chemical carcinogens,
$Risk_i$ = the risk estimate for the i^{th} chemical of n chemicals under evaluation.

This is conducted for each source of environmental release, associated exposure pathway, and receptor group at risk of exposure. Population risks may be derived by multiplying the overall risk level (summed for all subject chemicals) by the number of people exposed. This yields a measure of the additional incidence of developing cancer (i.e., additional number of new cases) in the exposed population over a lifetime (i.e., 70 years) of exposure. Population risk estimates were not developed for EFPC.

The traditionally accepted practice of evaluating exposure to noncarcinogenic compounds has been to experimentally determine a NOAEL and to divide this by a safety or uncertainty factor to establish an acceptable human dose or RfD (EPA 1989a). The RfD (or RfC for the inhalation route) is then compared to the average daily dose experienced by the exposed population to obtain a measure of concern for adverse noncarcinogenic effects:

$$HQ = \frac{Dose}{RfD} \tag{36}$$

where

HQ = hazard quotient: potential for adverse noncarcinogenic effects,
Dose = average daily dose for subchronic or chronic exposure [mg/(kg • d)],
RfD = acceptable intake for chronic exposure [mg/(kg • d)].

Dose and the RfD are expressed in the same units and are based upon common exposure periods (i.e., chronic, subchronic, or shorter-term). If the hazard quotient (HQ) is greater than 1, the potential may exist for adverse noncarcinogenic effects at the given exposure/dose level. Guidelines for evaluating exposure to mixtures of noncarcinogens are presented in EPA (1986b, 1989a). Essentially, this involves summing the HQ (ratios of daily dose/RfD or dose/RfC) for all chemicals under evaluation. If the sum of these ratios, called the hazard index (HI), is greater than 1, the potential exists for adverse noncarcinogenic effects.

$$HI = \sum_{j=1}^{n} HQ_j \tag{37}$$

Under these circumstances, EPA recommends summing HQ values for compounds with like or common toxicological effects, and evaluating the potential for manifestation of the various

adverse health effects. This is useful only if the HQs for individual substances are less than 1, but when combined sum to more than one.

5.4.2.2 Risk characterization methods for radionuclides

EPA Headquarters has identified two approaches for evaluating exposure to radionuclides at waste sites and the potential for carcinogenic effects (EPA 1989a). The first approach is based on the methods established by ICRP and is recommended as a basis for comparison with radiation protection criteria and standards or for evaluating risk in the occupational setting (EPA 1989a). The second approach, which was used at EFPC to estimate radionuclide risk, is advocated by EPA for evaluating risks to the general public.

The first approach is based on estimates of radiation dose equivalents. Dose equivalents to specified organs and the effective dose equivalent (to the whole body) are estimated. Intakes of radionuclides by the inhalation or ingestion routes are calculated by multiplying the amount of each radionuclide ingested or inhaled (i.e., expressed as activity) times an appropriate dose conversion factor:

$$Dose = C \times IF \times DCF \qquad (38)$$

where

Dose = effective whole body dose in rems,
C = concentration or activity of radionuclide in pCi/kg or pCi/L,
IF = intake of contaminated material for the exposure period of concern,
DCF = the dose conversion factor (rem/uCi) for the ingestion or inhalation routes.

Risk estimates are then derived using the following equation:

$$Risk = Dose \times CRF \qquad (39)$$

where

CRF = the cancer risk incidence factor.

The effective dose equivalent is a weighted sum of the dose equivalents to all irradiated organs and tissues and may be used as a measure of the overall detriment. The product of dose

equivalent (in Sieverts or rems) and a cancer incidence risk factor (in unit of risk per Sv or rem) yields an estimate of cancer risk incidence.

In RAGS (EPA 1989a), the Agency states that radiological risk estimates using this first approach are not applicable to the general public. Therefore, EPA advocates a second method for assessing radionuclide exposure to the general public. This approach uses CSFs that characterize the age-averaged lifetime excess cancer incidence per unit intake for each radionuclide of concern. The slope factors are based on age- and sex-specific coefficients for individual organs receiving radiation doses, and organ-specific dose conversion factors. Excess lifetime cancer risks are calculated as the product of the appropriate exposure point radionuclide activity administered to the receptor times the appropriate CSF:

$$Risk = A(t) \times CSF \tag{40}$$

where

A(t) = activity at a given time t in becquerel or picocuries (Bq or pCi),
CSF = route-specific CSF for the radionuclide (inverse Bq or pCi).

HEAST (EPA 1992e) provides CSFs for each of the four major pathways: inhalation, ingestion, air immersion, and ground-surface irradiation. The ingestion and inhalation slope factors have been used in this BRA; the air immersion and ground surface irradiation exposure pathways have been eliminated on the basis of site-specific information (as discussed in Sect. 5.1). Note that the focus of risk assessment for radionuclides is carcinogenicity. The evaluation of EFPC does not evaluate systemic toxicity following exposure to radioactive materials.

5.4.3 Tier I: Screening-level Risk Assessment

Comprehensive quantitative risk assessment is best conducted as a series of analyses progressing from a preliminary screening-level evaluation through increasing levels of refinement. As noted in Sect. 5.4.1, the human health risk assessment of EFPC has been conducted in three stages, or tiers. The first tier is an small-scale exploration of the potential risks to human health at three locations along EFPC. The results of this screening-level assessment are presented in this section. These results are used to obtain a first estimate of the magnitude of the potential risks to human health, and to help identify exposure pathways of importance and select COCs.

The Phase Ia sampling effort was based on historical records indicating that three locations (i.e., the NOAA site, the Bruner's site, and the Sturm property) should have some of the highest

levels of mercury contamination in the EFPC system. Data were collected from these three areas from three transects across EFPC (See Sects. 3 and 5.1 for a detailed discussion of this program). In the Phase Ia sampling program, data were collected on levels of contamination in floodplain soils, sediments, surface water, and groundwater. Soil data results were summarized for each of the three transects. Sediment, surface water, and groundwater data were summarized across transects (i.e., aggregated for all three locations). The risk assessment of the Phase Ia data focused on two receptor groups: adults and children ages 3 to 12 years. The following exposure pathways were included in the analysis:

- dermal exposure to surface water while swimming,
- dermal exposure to surface water while wading,
- incidental ingestion of surface water while swimming,
- dermal exposure to sediments while wading,
- dermal exposure to soil,
- incidental ingestion of soil,
- ingestion of produce grown on floodplain soils,
- ingestion of fish, and
- exposure to groundwater via ingestion and inhalation of vapors.

The focus of this first-tier assessment was to develop a conservative estimate of hypothetical long-term (chronic) exposure to contaminants detected at the three EFPC locations. In compliance with ORNL requests, a subchronic risk assessment also was included for young children (age 3) and adults.

The exposure assessment was based on use of RME (upper bound) and MLE (average or 50%) estimates. RME and MLE point estimates were adopted for environmental concentrations as well as for input variables used in the exposure assessment equations. The RME and MLE input variables are the same as those used in the agricultural/homesteader scenario, which was subsequently evaluated in the Tier II assessment. These exposure assumptions were chosen because they represent the most conservative exposure scenario. The Tier I screen was conducted prior to the Tier II and Tier III evaluations using toxicity values that were current at the time the screen was performed (October 1992). In keeping with EPA Region IV's request, only the RME risk estimates are presented in the risk characterization section. The results of both the RME and MLE evaluations are summarized in Appendix L.

5.4.3.1 Results of Tier I human health risk assessment

Hypothetical risks associated with long-term exposure to soil have been prepared for each of the Phase Ia sampling locations (i.e., NOAA, Bruner's, and Sturm). Results also have been summarized for the aggregated groundwater, surface water, and sediment data sets. The results of the Tier I assessment identified the following exposure pathways as principal concerns:

- ingestion exposure to produce from gardens grown in floodplain soils,
- inadvertent ingestion and dermal exposure to soils,
- ingestion exposure to groundwater, and
- ingestion exposure to fish.

Exposure to children and adults via these routes at each of the Tier I locations results in human health risk exceeding health risk targets defined by EPA.

The results of the Tier I assessment are based upon RME estimates, as summarized below. Appendix L provides both RME and MLE results because these two related point estimates more effectively assist in characterizing uncertainty.

Bruner's Site

Risks to human health are the greatest at the Bruner's site. The food chain pathways result in the greatest projected risk estimates. As discussed in the exposure section of the BRA (Sect. 5.2) and in the uncertainty analysis section (Sect. 5.5) that follows, these risks are attributable to the very conservative assumptions regarding contaminant partitioning from soil to plant tissues. Risk estimates for potential noncancer effects exceed an HI of 1.0 by more than an order of magnitude. The Tier I HI score for children ingesting produce is approximately 1×10^3, and 5×10^2 for adults. Cancer risk estimates for children and adults are 3×10^{-4} and 5×10^{-4}, respectively. The principal COCs for this pathway are arsenic, cadmium, manganese, mercury, and silver.

The Tier I assessment of inadvertent ingestion exposure to soils at the Bruner's site results in HI scores of 11 and 3 for children and adults, respectively. Cancer risk estimates are 5×10^{-5} for children and 4×10^{-5} for adults. The principal COC for this pathway is mercury. The risks for dermal exposure to soils fall within the target range established by EPA for waste site remediation.

Groundwater is not currently a source of drinking water for residents of the Oak Ridge community and is not anticipated to become a source of drinking water in the future. The State

of Tennessee has not yet formally classified the groundwater resource in the vicinity of EFPC with regard to its potential use as a source of drinking water. EPA specifies, however, that if a groundwater resource *may* be used as a source of drinking water (i.e., there are no constraints to this use), this pathway should be included in the BRA. For this reason, the groundwater pathways have been incorporated into the Tier I and Tier II assessments of EFPC.

The results of the risk assessment for hypothetical groundwater use fall outside the targets established by EPA for waste site remediation. The HI scores for both children and adults exceed 1.0 and the cancer risk estimates exceed 10^{-4}. In addition, the radionuclide risk for adults (2×10^{-4}) also exceed EPA's target cancer risk range of 10^{-6} to 10^{-4}. The key COCs are antimony, arsenic, beryllium, cadmium, manganese, mercury, PAHs, neptunium 237+D, and americium 241. No other media show risks that are above EPA-established guidelines.

NOAA Site

The cancer and noncancer risks at the NOAA site are lower than the Bruner's site, but follow the same pattern. Exposure from ingestion of produce is the pathway with the highest risk. The children and adult noncancer HIs (RME) are 7×10^{2} and 3×10^{2}, respectively. The excess lifetime cancer risks are 3×10^{-4} and 4×10^{-4}, respectively. All are above acceptable EPA levels and are due primarily to high risks from inorganic analytes (i.e., arsenic, cadmium, manganese, and mercury).

The soil ingestion pathway is also an exposure pathway of concern. Soil ingestion HIs of 8 (children) and 2 (adults) are due mainly to mercury contamination. Cancer risks (3×10^{-5} for both children and adults) fall within EPA's target cancer range. Risks from dermal contact with soils also fall within established guidelines.

Risks from exposure to surface water, sediments, groundwater, and fish are identical to those described for the Bruner's site, since these media were sampled and summarized across transects, not separately at each site.

Sturm Site

The Sturm site is the least contaminated of the three areas sampled for the Phase Ia evaluation. As with both of the other sites, the HIs and the cancer risks for the produce ingestion pathway exceed EPA guidelines. The children and adult HIs of 2×10^{2} and 80, respectively, are more than an order of magnitude greater than those from any other pathway. The cancer risks are 2×10^{-4} (children) and 3×10^{-4} (adults).

Cancer risks from ingestion and dermal contact with soil were within EPA's target risk range for both children and adults. The noncancer HI was above 1.0 only for a child ingesting soil. Risks from exposure to surface water, sediments, groundwater, and fish are the same as those described for the Bruner's site.

5.4.3.2 Evaluation of subchronic risks to children and adults

In compliance with requests from ORNL CRAC, an evaluation has been conducted of subchronic risks to the most sensitive receptor groups at EFPC. The receptor group is defined as children 3 years of age who may be exposed to contaminants originating from floodplain soils under agricultural land use conditions. There is currently no consensus in the risk assessment community as to how subchronic exposure should be precisely defined. EPA has stated in RAGS Vol. 1, Part B (EPA 1991c) that subchronic exposures occur over a period (duration) less than 7 years. EPA Region IV has not accepted this broad definition, preferring to limit subchronic exposures to 1 year. For the purposes of the Tier I assessment only, an exposure period of 1 year has been considered an appropriate timeframe (i.e., exposure duration) for subchronic exposure.

As it is inappropriate to evaluate subchronic risks for carcinogens, only noncarcinogenic effects have been carried through this subchronic evaluation. In addition, HIs have been calculated only for exposures at the Bruner's site (because this site is the most contaminated, and therefore shows the highest risks), and only using RME point estimates. The results of this subchronic evaluation are presented along with other Tier I results in Appendix L.

HIs are the highest for the food chain pathways. Exposure from ingestion of produce results in an HI of 4×10^3 for children age 3 years. Exposure to beef and dairy products also shows unacceptable HIs at 3×10^2 and 4×10^2 for children. The HIs associated with the fish ingestion pathway are below the HI score of 1.0. Two other pathways, soil ingestion and groundwater ingestion, show HIs above 1.0. These HIs are 18 and 8, respectively.

The magnitude of the HI scores for subchronic effects is driven by elevated levels of mercury. For the groundwater and food chain pathways, exposures to other inorganic contaminants, including arsenic and manganese, also contribute to the high HIs.

5.4.3.3 Summary of the screening-level analysis

The results of the Tier I screening-level human health risk assessment have been used to explore exposure pathways and to assist in selecting COCs. These results have been used in refining the approach to the creek-wide human health risk assessment (Tier II) that is based on

use of the full monitoring data set (i.e., Phases Ia and Ib). From the results of the Tier I assessment, mercury is confirmed as the primary COC and a principal driver of the observed risks to human health.

5.4.4 Tier II: Human Health Risk Assessment Based on Phase Ia and Ib Data

The Tier II BRA is a creek-wide evaluation of the potential risks to human health. This assessment makes use of an integrated data set, including monitoring data from Phases Ia and Ib. The risk assessment has been conducted as a function of creek segment and land use area, and examines potential risks to adults and children under current and future use. A detailed overview of creek segmentation, and the land use scenarios and exposure pathways of concern, is presented in Sects. 5.1 and 5.2. It is important to recognize that the results of risk assessment may only be understood in light of the assumptions that form the basis of the risk estimates.

Although a given segment may be characterized by multiple land uses, it is land use within the EFPC 100-year floodplain area that is the primary focus of the "current land use" risk assessment. The evaluation of future use incorporates risk assessment of likely (projected) land use scenarios. An additional analysis is included for open land use in three areas of elevated contamination along the SLB.

EFPC is a large study area and the human health risk assessment has required the organization and manipulation of a large quantity of information. An integrated spreadsheet model has been developed incorporating data on exposure point concentrations, exposure assumptions, contaminant partitioning between environmental and biological media, and toxicity measures. The model is used to generate both deterministic estimates (i.e., point estimates) and probabilistic risk estimates (i.e., use of Monte Carlo analysis). Risk estimates based on RME assumptions provide the conservative, single-point determinations specified by EPA Headquarters and requested by EPA Region IV. The results of the Monte Carlo assessment (presented in the Tier III risk assessment in Sect. 5.5) supplement the RME point estimates. Monte Carlo results are probabilistic estimates of potential risk that make explicit the uncertainty and variability surrounding the point estimates based on RME assumptions.

The results of the creek-wide risk assessment (Tier II) are presented in several complementary ways. Separate risk characterization tables have been prepared for each exposure pathway under evaluation, in each of the nine creek segments. The tables include each receptor group (i.e., adults and children) and each land use area under evaluation, for both current and future land use. The tables present the following information:

- chemical name,
- exposure point concentrations,

- chronic daily intake for noncancer exposure estimates,
- chronic daily intake for cancer risk estimates,
- HQs and HIs,
- principal noncarcinogenic effects of concern,
- uncertainty factors used in derivation of the RfD or RfC,
- cancer risk estimates, and
- EPA weight-of-evidence ratings for each carcinogen.

These detailed tables show the MLE and RME noncancer and cancer risk estimates for each exposure route (i.e., ingestion, dermal, and inhalation) and each analyte, and provide the related exposure point concentration, the principal noncancer health effect, and the cancer weight-of-evidence. There are literally hundreds of these tables, so in an effort to ensure a coherent presentation of results, these tables are included in this report as a group in Appendix M.

From the detailed risk characterization tables for each pathway (in Appendix M), RME summary tables also have been prepared and are presented for each creek segment and land use area. Tables 5.20 through 5.41, presented in Sects. 5.4.4.2 and 5.4.4.3, summarize the RME results of the BRA, for each relevant exposure scenario, and for both current and future land uses. The summary tables provide an overview and improve understanding of the many risk estimates. The summary risk estimates are presented along with a letter designator, to specify whether the risks exceed targets established by EPA for waste site remediation under the Superfund program (EPA 1989a). The designators are as follows:

- B = HI is below 1.0 — also for excess lifetime cancer risk below 10^{-6},
- W = incremental lifetime cancer risk falls within the range of 10^{-6} to 10^{-4}, and
- E = HI exceeds 1.0 — also for excess lifetime cancer risk exceeding 10^{-4}.

The summary tables present the results of the risk assessment for each pathway and route of exposure, for a given receptor (i.e., adults or children) and exposure scenario (e.g., residential land use). Current and future risks are discussed separately for land use areas in each of the nine segments designated for EFPC. For each receptor, estimates are presented separately for both noncancer effects (HIs) and excess lifetime cancer risks. The noncancer and cancer risk estimates are further grouped by exposure route (i.e., ingestion, dermal, and inhalation), and these are reported for each medium. The cancer risks associated with exposure to nonradionuclide substances are distinguished from those related to radionuclide exposures.

In the discussion of Tier II results, the following approach is used. *First*, current results are considered separately from future results for a particular segment. *Second*, noncancer risk

estimates (HQs and HIs) are noted, along with the key substances contributing to noncancer effects. Key substances are those with an HQ of 1 or greater, or several substances with HQs that sum to an HI greater than 1. *Third*, cancer risk estimates are noted, along with the key substances contributing to the cancer risk estimate. This is most important for substances with individual cancer risks greater than 1×10^{-4}. In addition, individual substances with cancer risks greater than 1×10^{-6} are discussed, especially if the sum of several of these substances approaches or exceeds 1×10^{-4}. Substances with associated cancer risks of less than 1×10^{-6} are not discussed in the text, but are indicated in the detailed tables in Appendix M. *Finally*, a list of the primary substances of concern for each segment (or area) are noted along with an indication of environmental media of importance (i.e., soil, groundwater, produce). These appear in a list at the end of the subsection for each segment, and only appear if there are substances that individually or when combined result in risk estimates that exceed EPA targets for waste site remediation.

Risk characterization of lead exposures is evaluated using a method that differs from that used for the other chemical and radionuclide substances: EPA lead biokinetic model. Therefore, a separate portion of the Tier II risk characterization is presented for the discussion of lead. The required elements for the lead risk characterization, such as selection of exposure point concentrations, the method used to estimate risk, and the results, are all presented in the lead section to maintain clarity.

5.4.4.1 Results of Tier II human health risk assessment

Before reviewing the results of the risk assessment, several features of the risk characterization must be discussed. It is important to re-emphasize that the human health risk estimates for EFPC are based upon the assumptions of the human exposure assessment, and are not measures of actual exposure. The results are analytical tools for decisionmaking and provide information regarding the significance of the observed levels of contamination.

Estimates of combined risk for a given receptor should be based on a reasonable combination of exposures across pathways. In this risk assessment, the summary risk estimates are shown both uncombined and combined to provide analytical flexibility. Risks are presented for each exposure pathway and exposure route, and are shown summed across all exposure routes as a very conservative estimate of total site risk. Combining all such exposures for the total site risk estimate is very conservative. The probability of exposure events occurring to an individual receptor becomes more remote as each event is included, and the sum total tends to overestimate likely exposures.

Groundwater, surface water, and sediment exposures were based on a site-wide aggregation of data, which was selected for use in the EFPC BRA by consensus with EPA Region IV, DOE, and ORNL CRAC. In using this approach, the exposure point concentrations for these three media are identical between segments.

Exposure to groundwater is based on the results of unfiltered samples. Although the unfiltered water samples were taken from properly developed monitoring wells, the levels of suspended particulates in the monitoring well water are likely to be greater than water from an active drinking water supply well. With the use of unfiltered groundwater samples, the groundwater risk estimates are very conservative and overestimate risks at a hypothetical drinking water supply well. A supplemental risk assessment was conducted as a point of comparison using the filtered groundwater samples.

The additional risk characterization of the SLB is limited to soil exposures only. The areas under consideration along the SLB are limited to open land use, and no exposures to groundwater, sediments, or other media are likely.

Background levels of contaminants have not been subtracted out before determining exposure point concentrations and evaluating risks to human health. In keeping with EPA guidance in RAGS Vol. 1, Part A (EPA 1989a), the risk estimates presented in this section reflect exposure to both site-related and background levels. The influence of observed background concentrations is subsequently evaluated. Background soil samples have been obtained from the Hinds Creek area, a location remote to the DOE Y-12 Plant and EFPC. Two methods have been used to evaluate the significance of background concentrations in the BRA of EFPC:

- comparison of data from the EFPC floodplain to that of the Hinds Creek background location using a tolerance interval approach, and
- a supplemental risk assessment has been conducted using background data from the Hinds Creek-location and exposure assumptions for residential land use.

The tolerance interval approach has been adopted as a basis for statistically comparing contaminant levels in each land use segment with concentrations observed at the Hinds Creek background locations. The significance of background levels is evaluated by determining the proportion of samples for each COC in EFPC floodplain soils that exceed the 95th percentile tolerance interval of the background data set (i.e., from Hinds Creek soils). This evaluation is presented in Sect. 5.4.3.2. The summary tables showing the tolerance interval statistics for each land use segment are presented in Appendix P. The supplemental risk assessment based on soil data from Hinds Creek is presented at the end in Sect. 5.4.3.3. As a point of comparison, risk

assessment also was conducted for the groundwater ingestion pathway using filtered groundwater samples.

5.4.4.2 Current land use conditions

This section presents the results of the Tier II risk assessment for current land use along EFPC. In compliance with a request from EPA Region IV, only the RME risk estimates are presented and evaluated in this section. Risk estimates are provided for noncarcinogenic and carcinogenic effects, for adults and children, for each land use area, and within each of the designated segments along EFPC.

SEGMENT 1 - CURRENT

Land use along the EFPC floodplain at Segment 1 is currently limited to the open designation. Currently, no risks related to commercial land use are considered likely within the sampled portions of Segment 1. None of the HIs or incremental lifetime cancer risk estimates related to open land use exceeds the target risk ranges established by EPA for waste site remediation under the Superfund program. Summary risk estimates for exposures associated with current land use at Segment 1 are presented in Table 5.20.

Noncancer Effects

Open Land Use. Table 5.20 indicates that the highest HI score, combining exposure across soil, surface water, and sediment pathways, falls below the target (i.e., less than 1) established by EPA.

Cancer Effects

Open Land Use. The cancer risks attributable to the chemical carcinogens dominate, and are more than an order of magnitude greater than the radionuclide cancer risks. However, all of the risks are within the noted target cancer risk range established by EPA.

Cancer risks for soil ingestion of chemical carcinogens are on the order of 10^{-5}, and account for more than one-half of the total cancer risk estimate. The risk estimates for dermal contact with soil are less, on the order of 10^{-6}. Three substances are responsible for the bulk of the total soil ingestion cancer risk estimate: arsenic (26%), Aroclor-1260 (26%), and benzo(a)pyrene (17%). The combined excess lifetime cancer risk estimates for radionuclides are below the EPA target, at 1×10^{-7}.

Table 5.20. Risk characterization summary for EFPC Segment 1: RME estimates for current land use

		AGRICULTURAL LAND USE						COMMERCIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index: Combined Exposure		NA	NA					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	NA

		OPEN LAND USE						RESIDENTIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	7.37E−01 B	3.88E−01 B	6.52E−06 W	1.14E−05 W	1.16E−07 B	2.90E−07 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	4.53E−02 B	4.21E−02 B	2.53E−06 W	7.84E−06 W	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	1.48E−03 B	1.04E−03 B	3.10E−09 B	7.22E−09 B	4.89E−10 B	1.63E−09 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	1.28E−03 B	1.18E−03 B	3.01E−08 B	9.31E−08 B	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	9.90E−05 B	9.19E−05 B	1.26E−08 B	3.89E−08 B	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	--	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	--	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	--	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	--	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	--	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	--	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	--	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index: Combined Exposure		7.9E−01 B	4.3E−01 B					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				9E−06 W	2E−05 W					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						1E−07 B	3E−07 B					NA	NA

B – below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1 x 10^{-6})
W – within EPA target cancer risk range (ELCR> 1 x 10^{-6} and < 1 x 10^{-4})
B – exceeds EPA target for noncancer hazard index (HI>1), or cancer risk (ELCR< 1 x 10^{-4})

SEGMENT 2 - CURRENT

As noted in Sect. 5.2.1, current land use in the immediate vicinity of Segment 2 includes residential, commercial, and open land use. However, the entire sampled portion of Segment 2 is currently limited to open land use, and no commercial risks are considered likely. None of the noncancer HIs or cancer risks for current open land use at Segment 2 exceeds EPA targets, although cancer risks are within the target range specified by EPA for waste site remediation. Table 5.21 presents summary risk estimates for exposures associated with current land use at Segment 2.

Noncancer Effects

Open Land Use. The combined exposure HI for current open land use at Segment 2 is below the EPA noncancer effects target of 1.

Cancer effects

Open Land Use. At Segment 2, nonradionuclide cancer risks for both adults and children fall within EPA's target cancer risk range. Soil ingestion and dermal contact outweigh the other exposure pathways and routes, and of these two, soil ingestion is the most important. Four substances have independent soil ingestion cancer risks greater than 10^{-6}: arsenic with 41%, beryllium with 11%, Aroclor-1260 with 12%, and benzo(a)pyrene with 19% of the soil ingestion cancer risk. The remaining substances have independent cancer risks on the order of 10^{-7} or less. Radionuclide cancer risks are all below the EPA target cancer risk range.

SEGMENT 3 - CURRENT

The sampled portion of Segment 3 includes residential, commercial, and open land uses, although no commercial risks are currently considered likely. At Segment 3, none of the noncancer and cancer risk estimates for current open land use exceeds EPA targets. However, for residential land use, both HIs and cancer risks exceed the EPA-established guidelines. Summary risk estimates for current land use at Segment 3 are presented in Table 5.22.

Noncancer Effects

Open Land Use. The combined exposure HIs for current open land use at Segment 3 are all below the EPA noncancer effects limit of 1.

Residential Land Use. The combined exposure HIs for current residential land use exceed the EPA target for noncancer effects, and these are overwhelmingly attributable to the

Table 5.21. Risk characterization summary for EFPC Segment 2: RME estimates for current land use

		AGRICULTURAL LAND USE						COMMERCIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards:													
Hazard Index:		NA	NA					NA	NA				
Combined Exposure													
Excess Lifetime Cancer Risk:				NA	NA					NA	NA		
Combined Exposure													
Radiological Hazards:													
Excess Lifetime Cancer Risk:						NA	NA					NA	NA
Combined Exposure													

		OPEN LAND USE						RESIDENTIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	1.95E-01 B	1.03E-01 B	5.91E-06 W	1.04E-05 W	7.77E-08 B	1.95E-07 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	1.21E-02 B	1.13E-02 B	1.90E-06 W	5.89E-06 W	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	1.48E-03 B	1.04E-03 B	3.10E-09 B	7.22E-09 B	4.89E-10 B	1.63E-09 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	1.28E-03 B	1.18E-03 B	3.01E-08 B	9.31E-08 B	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	9.90E-05 B	9.19E-05 B	1.26E-08 B	3.89E-08 B	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards:													
Hazard Index:		2.1E-01 B	1.2E-01 B					NA	NA				
Combined Exposure													
Excess Lifetime Cancer Risk:				8E-06 W	2E-05 W					NA	NA		
Combined Exposure													
Radiological Hazards:													
Excess Lifetime Cancer Risk:						8E-08 B	2E-07 B					NA	NA
Combined Exposure													

B – below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1 x 10^{-6})
W – within EPA target cancer risk range (ELCR>1 x 10^{-6} and <1 x 10^{-4})
B – exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1 x 10^{-4})

Table 5.22. Risk characterization summary for EFPC Segment 3: RME estimates for current land use

MEDIA	ROUTE	AGRICULTURAL LAND USE: NONCANCER CHILD	AGRICULTURAL LAND USE: NONCANCER ADULT	AGRICULTURAL LAND USE: CANCER CHILD	AGRICULTURAL LAND USE: CANCER ADULT	AGRICULTURAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	AGRICULTURAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT	COMMERCIAL LAND USE: NONCANCER CHILD	COMMERCIAL LAND USE: NONCANCER ADULT	COMMERCIAL LAND USE: CANCER CHILD	COMMERCIAL LAND USE: CANCER ADULT	COMMERCIAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	COMMERCIAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards:													
Hazard Index (HI): Combined Exposure		NA	NA					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	NA		
Radiological Hazards:													
Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	NA

MEDIA	ROUTE	OPEN LAND USE: NONCANCER CHILD	OPEN LAND USE: NONCANCER ADULT	OPEN LAND USE: CANCER CHILD	OPEN LAND USE: CANCER ADULT	OPEN LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	OPEN LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT	RESIDENTIAL LAND USE: NONCANCER CHILD	RESIDENTIAL LAND USE: NONCANCER ADULT	RESIDENTIAL LAND USE: CANCER CHILD	RESIDENTIAL LAND USE: CANCER ADULT	RESIDENTIAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	RESIDENTIAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT
Soil	Ingestion	2.80E−01 B	1.47E−01 B	5.80E−06 W	1.02E−05 W	6.95E−08 B	1.74E−07 B	1.23E+00 *B*	3.44E−01 B	2.55E−05 W	2.37E−05 W	1.62E−07 B	4.06E−07 B
	Dermal Contact	1.74E−02 B	1.61E−02 B	1.97E−06 W	6.10E−06 W	NA	NA	4.92E−02 B	3.76E−02 B	5.58E−06 W	1.42E−05 W	NA	NA
Surface Water	Ingestion	1.48E−03 B	1.04E−03 B	3.10E−09 B	7.22E−09 B	4.89E−10 B	1.63E−09 B	2.32E−02 B	8.62E−03 B	4.85E−08 B	6.01E−08 B	4.07E−09 B	1.36E−08 B
	Dermal Contact	1.28E−03 B	1.18E−03 B	3.01E−08 B	9.31E−08 B	NA	NA	1.73E−02 B	1.32E−02 B	4.08E−07 B	1.04E−06 W	NA	NA
Sediment	Dermal Contact	9.90E−05 B	9.19E−05 B	1.26E−08 B	3.89E−08 B	NA	NA	8.66E−04 B	6.62E−04 B	1.10E−07 B	2.80E−07 B	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	9.83E+01 *B*	4.74E+01 *B*	2.57E−04 *B*	4.13E−04 *B*	1.65E−07 B	7.13E−07 B
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	2.01E−01 B	9.63E−02 B	4.68E−05 W	7.47E−05 W	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards:													
Hazard Index (HI): Combined Exposure		3.0E−01 B	1.7E−01 B					1.0E+02 *B*	4.8E+01 *B*				
Excess Lifetime Cancer Risk: Combined Exposure				8E−06 W	2E−05 W					3E−04 *B*	5E−04 *B*		
Radiological Hazards:													
Excess Lifetime Cancer Risk: Combined Exposure						7E−08 B	2E−07 B					3E−07 B	1E−06 W

B – below or equal to target noncancer hazard index (HI<1), or cancer risk (ELCR<=1 x 10^{-6})
W – within EPA target cancer risk range (ELCR>1 x 10^{-6} and <1 x 10^{-4})
B – exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1 x 10^{-4})

produce ingestion pathway. The only other significant HIs are for the soil ingestion pathway, with HIs of 1.2 (children) and 0.3 (adults). The HI of 1.2 is at the limit of the EPA target, when rounded to one significant figure. The produce ingestion HIs are well above the acceptable noncancer HI limit of 1. Although the soil ingestion HI for children is at the limit of the EPA target, the corresponding MLE is well below the target, at 0.3. Substances of concern for the produce ingestion pathway include mercury with 87%, manganese with 5%, arsenic with 4%, and cadmium with 3% of the produce ingestion HI.

The fish ingestion HIs are 0.2 (children) and 0.1 (adults), which are below the target HI limit of 1. The remaining exposures are relatively minor with much lower combined exposure HIs for both children and adults.

Cancer Effects

Open Land Use. For current open land use, the summed nonradionuclide cancer risks at Segment 3 are all within the EPA target cancer risk range. Cancer risks related to radionuclide substances are even lower.

None of the cancer risks for current open land use at Segment 3 exceeds EPA targets. Soil ingestion outweighs the soil dermal contact pathway, and substances of concern for the soil ingestion pathway are arsenic with 41%, beryllium with 8%, Aroclor-1260 with 6%, benzo(a)pyrene with 22%, and dibenzo(a,h)anthracene with 7% of the soil ingestion cancer risk. The major substance of concern for the soil dermal contact pathway is benzo(a)pyrene, with 39% of the soil dermal contact cancer risk.

All of the radionuclide cancer risks for current open land use at Segment 3 are minor, and are all below the EPA target cancer range.

Residential Land Use. For current residential land use at Segment 3, the total cancer risk is predominated by the nonradionuclide exposures, and these exceed the EPA cancer risk range target.

The residential produce ingestion pathway contributes the greatest to the nonradionuclide cancer risk, with risks of 3×10^{-4} (children) and 4×10^{-4} (adults). The remaining risks are primarily from the fish ingestion, soil ingestion, and soil dermal contact pathways. Cancer risks for each of these are within the acceptable EPA cancer risk range of 10^{-6} to 10^{-4}.

The radionuclide cancer risk is relatively minor, with a combined total of 1 x 10^{-6} for the adult resident. This is at the lower limit of the EPA target cancer risk range.

The main substance of concern for the produce ingestion pathway is arsenic, representing 97% of the cancer risk. Beryllium, benzo(a)anthracene, benzo(a)pyrene, and dibenzo(a,h)-anthracene each have independent cancer risks on the order of 10^{-6}. The two Aroclors represent all of the cancer risks related to the fish ingestion pathway..

Two substances have soil ingestion cancer risks exceeding 1 $\times$ 10^{-6}. Arsenic accounts for 41% and benzo(a)pyrene accounts for 22% of the soil ingestion cancer risk. Only benzo(a)pyrene has a soil dermal contact cancer risk greater than 1 $\times$ 10^{-6}, representing 39% of the soil dermal contact cancer risk.

Primary Substances of Concern

- Arsenic (produce)
- Cadmium (produce)
- Manganese (produce)
- Mercury (produce).

SEGMENT 4 - CURRENT

As noted in Sect. 5.2.1, half of Segment 4 is currently used for residential purposes, and the remaining half is divided between commercial and open land use. However, the sampled portion of Segment 4 is entirely under open land use. All of the noncancer and cancer risks for current open land use at Segment 4 are below EPA targets. Table 5.23 presents summary risk estimates for exposures associated with current land use at Segment 4.

Noncancer Effects

Open Land Use. The combined exposure HI for open land use at Segment 4 is below the EPA target noncancer effects limit of 1.

Cancer Effects

Open Land Use. At Segment 4, nonradionuclide risks fall within the EPA target cancer risk range. These risks are almost entirely attributable to the soil contact pathways. Soil ingestion cancer risks are slightly greater than the dermal contact route. Radionuclide cancer risks for current open land use at Segment 4 are all well below the EPA target cancer risk range.

Table 5.23. Risk characterization summary for EFPC Segment 4: RME estimates for the current land uses

		AGRICULTURAL LAND USE						COMMERCIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		NA	NA					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	NA

		OPEN LAND USE						RESIDENTIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	8.18E−01 B	4.31E−01 B	8.99E−06 W	1.58E−05 W	8.39E−08 B	2.10E−07 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	5.02E−02 B	4.66E−02 B	3.51E−06 W	1.09E−05 W	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	1.48E−03 B	1.04E−03 B	3.10E−09 B	7.22E−09 B	4.89E−10 B	1.63E−09 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	1.19E−03 B	1.11E−03 B	3.01E−08 B	9.31E−08 B	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	9.78E−05 B	9.19E−05 B	1.26E−08 B	3.89E−08 B	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		8.7E−01 B	4.8E−01 B					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				1E−05 W	3E−05 W					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						8E−08 B	2E−07 B					NA	NA

B – below or equal to limit of noncancer hazard index (HI<=1), or cancer risk (ELCR<=1×10^{-6})
W – within EPA target cancer risk range (ELCR>1×10^{-6} and <1×10^{-4})
B – exceeds EPA target for noncancer hazard index (HI>1), or cancer risk (ELCR>1×10^{-4})

Substances with individual cancer risks greater than 1×10^{-6} include arsenic, beryllium, Aroclor-1254, Aroclor-1260, and benzo(a)pyrene.

SEGMENT 5 - CURRENT

The sampled portion of Segment 5 is entirely under the open land use designation, and no commercial risks are currently considered likely within the sampled portions of this segment. None of the noncancer HIs or cancer risks for current open land use at Segment 5 exceeds EPA targets. Summary risk estimates for exposures associated with current land use at Segment 5 are presented in Table 5.24.

Noncancer Effects

Open Land Use. The combined exposure HIs for open land use at Segment 5 are all below the EPA target noncancer effects limit of 1.

Cancer Effects

Open Land Use. The summed nonradionuclide and radionuclide cancer risks are predominated by the nonradionuclide exposures, which are within the EPA target cancer risk range. Radionuclide cancer risks for current open land use at Segment 5 are lower than the nonradionuclide risks and also are below the EPA target cancer risk range.

The soil ingestion and dermal pathways are the only important contributors to total cancer risk. The soil ingestion cancer risks of arsenic and benzo(a)pyrene are greater than 1×10^{-6}. For the dermal route, seven substances, all with cancer risks on the order of 10^{-7}, combine to a cancer risk greater than 1×10^{-6}.

SEGMENT 6 - CURRENT

The sampled portion of Segment 6 is entirely under residential land use, and risk estimates have been calculated only for the residential land use pathways. Table 5.25 presents summary risk estimates for exposures associated with current land use at Segment 6.

Noncancer Effects

Residential Land Use. The combined exposure HIs for residential land use at Segment 6 are well above the EPA noncancer target. These HIs are primarily due to produce ingestion. Other significant pathways include creek fish ingestion, soil ingestion, and soil dermal contact, and have a combined HI that is below the target noncancer effects limit of 1.

Table 5.24. Risk characterization summary for EFPC Segment 5: RME risk estimates for current land uses

		AGRICULTURAL LAND USE						COMMERCIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		NA	NA					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	NA

		OPEN LAND USE						RESIDENTIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	2.81E-01 B	1.48E-01 B	4.52E-06 W	7.93E-06 W	5.01E-08 B	1.26E-07 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	1.73E-02 B	1.61E-02 B	1.47E-06 W	4.54E-06 W	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	1.48E-03 B	1.04E-03 B	3.10E-09 B	7.22E-09 B	4.89E-10 B	1.63E-09 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	1.28E-03 B	1.18E-03 B	3.01E-08 B	9.31E-08 B	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	9.90E-05 B	9.19E-05 B	1.26E-08 B	3.89E-08 B	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		3.0E-01 B	1.7E-01 B					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				6E-06 W	1E-05 W					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						5E-08 B	1E-07 B					NA	NA

B – below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1 x 10^{-6})
W – within EPA target cancer risk range (ELCR>1 x 10^{-6} and <1 x 10^{-4})
B – exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1 x 10^{-4})

Table 5.25. Risk characterization summary for EFPC Segment 6: RME estimates for current land uses

		AGRICULTURAL LAND USE						COMMERCIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards:													
Hazard Index (HI): Combined Exposure		NA	NA					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	NA		
Radiological Hazards:													
Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	NA

		OPEN LAND USE						RESIDENTIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	6.69E-01 B	1.87E-01 B	1.32E-05 W	1.23E-05 W	2.30E-07 B	5.76E-07 B
	Dermal Contact	NA	NA	NA	NA	NA	NA	2.65E-02 B	2.03E-02 B	1.65E-06 W	4.21E-06 W	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	2.32E-02 B	8.62E-03 B	4.85E-08 B	6.01E-08 B	4.07E-09 B	1.36E-08 B
	Dermal Contact	NA	NA	NA	NA	NA	NA	1.73E-02 B	1.32E-02 B	4.08E-07 B	1.04E-06 W	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	8.66E-04 B	6.62E-04 B	1.10E-07 B	2.80E-07 B	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	5.05E+01 *B*	2.44E+01 *B*	1.97E-04 *B*	3.16E-04 *B*	1.76E-07 B	7.60E-07 B
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	2.01E-01 B	9.63E-02 B	4.68E-05 W	7.47E-05 W	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards:													
Hazard Index (HI): Combined Exposure		NA	NA					5.1E+01 *B*	2.5E+01 *B*				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					3E-04 *B*	4E-04 *B*		
Radiological Hazards:													
Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					4E-07 B	1E-06 W

B – below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1 x 10^{-6})
W – within EPA target cancer risk range (ELCR>1 x 10^{-6} and <1 x 10^{-4})
B – exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1 x 10^{-4})

Cancer Effects

Residential Land Use. The nonradionuclide cancer risks are greater than the EPA target cancer risk range, and are mostly attributable to the produce ingestion pathway. Radionuclide cancer risks are two orders of magnitude less, with a combined cancer risk for adults at the lower limit of the EPA cancer risk target (1×10^{-6}).

The greatest nonradionuclide cancer risks for current residential land use at Segment 6 are for produce ingestion, which exceed the EPA cancer risk target. Additional cancer risks attributable to fish ingestion and soil ingestion are on the order of 10^{-5}, which is within the EPA target range. The remaining pathways are relatively minor, with surface water ingestion, and soil, sediment, and surface water dermal contact pathways combining to yield a cancer risk of 3×10^{-6}.

Substances of primary concern for the produce ingestion pathway are arsenic, which represents 98% of the cancer risk, and beryllium. Only the cancer risk for arsenic is in excess of the EPA cancer risk target. For the fish ingestion pathway, the two Aroclor congeners represent all of the cancer risk. The soil ingestion cancer risk is spread across 10 carcinogenic analytes, which each have independent cancer risks of similar magnitude.

Primary Substances of Concern

- Arsenic (produce)
- Cadmium (produce)
- Manganese (produce)
- Mercury (produce).

SEGMENT 7 - CURRENT

As noted in Sect. 5.2.1, Segment 7 currently falls under the agricultural and open land uses. Summary risk estimates for exposures associated with current land use at Segment 7 are presented in Table 5.26.

Noncancer Effects

Open Land Use. The combined HIs for open land use at Segment 7 are all below the EPA limit for noncancer effects.

Agricultural Land Use. The HIs for agricultural land use at Segment 7 are well above the EPA-established noncancer guideline of 1. This is almost entirely due to the produce and beef

Table 5.26. Risk characterization summary for EFPC Segment 7: RME risk estimates for current land uses

MEDIA	ROUTE	AGRICULTURAL LAND USE: NONCANCER CHILD	AGRICULTURAL LAND USE: NONCANCER ADULT	AGRICULTURAL LAND USE: CANCER CHILD	AGRICULTURAL LAND USE: CANCER ADULT	AGRICULTURAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	AGRICULTURAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT	COMMERCIAL LAND USE: NONCANCER CHILD	COMMERCIAL LAND USE: NONCANCER ADULT	COMMERCIAL LAND USE: CANCER CHILD	COMMERCIAL LAND USE: CANCER ADULT	COMMERCIAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	COMMERCIAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT
Soil	Ingestion	5.34E-01 B	1.49E-01 B	1.41E-05 W	1.32E-05 W	2.17E-07 B	5.43E-07 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	2.12E-02 B	1.62E-02 B	1.64E-06 W	4.19E-06 W	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	2.32E-02 B	8.62E-03 B	4.85E-08 B	6.01E-08 B	4.07E-09 B	1.36E-08 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	1.73E-02 B	1.32E-02 B	4.08E-07 B	1.04E-06 W	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	8.66E-04 B	6.62E-04 B	1.10E-07 B	2.80E-07 B	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	9.04E+01 *B*	4.36E+01 *B*	4.92E-04 *B*	7.91E-04 *B*	7.16E-07 B	3.10E-06 W	NA	NA	NA	NA	NA	NA
Beef	Ingestion	2.01E+01 *B*	1.01E+01 *B*	1.66E-04 *B*	2.79E-04 *B*	2.21E-06 W	9.99E-06 W	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	2.01E-01 B	9.63E-02 B	4.68E-05 W	7.47E-05 W	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	2.34E-01	NA	2.55E-06	NA	8.96E-06	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		1.1E+02 *B*	5.4E+01 *B*					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				7E-04 *B*	1E-03 *B*					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						3E-06 W	2E-05 W					NA	NA

MEDIA	ROUTE	OPEN LAND USE: NONCANCER CHILD	OPEN LAND USE: NONCANCER ADULT	OPEN LAND USE: CANCER CHILD	OPEN LAND USE: CANCER ADULT	OPEN LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	OPEN LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT	RESIDENTIAL LAND USE: NONCANCER CHILD	RESIDENTIAL LAND USE: NONCANCER ADULT	RESIDENTIAL LAND USE: CANCER CHILD	RESIDENTIAL LAND USE: CANCER ADULT	RESIDENTIAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	RESIDENTIAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT
Soil	Ingestion	1.21E-01 B	6.39E-02 B	3.21E-06 W	5.64E-06 W	9.29E-08 B	2.33E-07 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	7.49E-03 B	6.96E-03 B	5.80E-07 B	1.80E-06 W	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	1.48E-03 B	1.04E-03 B	3.10E-09 B	7.22E-09 B	4.89E-10 B	1.63E-09 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	1.28E-03 B	1.18E-03 B	3.01E-08 B	9.31E-08 B	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	9.90E-05 B	9.19E-05 B	1.26E-08 B	3.89E-08 B	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		1.3E-01 B	7.3E-02 B					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				4E-06 W	8E-06 W					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						9E-08 B	2E-07 B					NA	NA

B – below or equal to target noncancer hazard index (HI <=1), or cancer risk (ELCR <= 1×10^{-6})
W – within EPA target cancer risk range (ELCR > 1×10^{-6} and < 1×10^{-4})
B – exceeds target noncancer hazard index (HI > 1), or cancer risk (ELCR > 1×10^{-4})

ingestion pathways. These are the only pathways with HIs greater than the EPA target for noncancer effects. For produce ingestion, four substances are responsible for the majority of the HI: arsenic, cadmium, manganese, and mercury. For beef ingestion, four substances have HQs greater than 1: arsenic, mercury, selenium, and zinc.

Cancer Effects

Open Land Use. The combined nonradionuclide cancer risks for open land use at Segment 7 are within the target cancer risk range established by EPA. The most important exposure pathways responsible for these risks are soil ingestion and soil dermal contact. The primary substance of concern for cancer risk is arsenic. All remaining carcinogens have independent cancer risks on the order of 10^{-7} or less. The radionuclide cancer risks are below the EPA target cancer risk range.

Agricultural Land Use. Food chain exposures for the agricultural scenario at Segment 7 include produce, beef, and fish ingestion, but exclude dairy ingestion. The combined cancer risks for produce and beef ingestion exceed the EPA target cancer risk range, although the risks for fish ingestion do not. Also falling within the cancer risk range are risks for the soil ingestion and soil dermal contact pathways. The total cancer risk estimates for the radionuclides are approximately two orders of magnitude less than the nonradionuclides.

The primary nonradionuclide substance of carcinogenic concern for produce ingestion is arsenic, which accounts for 98% of the total produce ingestion cancer risk. The cancer risk for arsenic exceeds the EPA target, although the cancer risks for beryllium and benzo(a)pyrene are within the target range. The greatest radionuclide cancer risks for produce ingestion are on the order of 10^{-6}, which is within the EPA target range for cancer effects. The remaining nonradionuclide and radionuclide substance cancer risks are less than 1×10^{-6}.

For beef ingestion, only arsenic has a cancer risk exceeding the upper limit of the EPA target cancer risk range (1×10^{-4}). Arsenic represents 49% of the total beef ingestion cancer risk. Substances with cancer risks greater than 1×10^{-6} include: beryllium, Aroclor-1260, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, and indeno(1,2,3-cd)pyrene. These substances represent nearly all the remaining cancer risk. Radionuclide substances with beef ingestion cancer risks greater than 1×10^{-6} include thorium-230 and thorium-232.

For the fish ingestion pathway, Aroclor-1254 and Aroclor-1260 represent all of the fish ingestion cancer risk.

Primary Substances of Concern

- Arsenic (produce and beef)
- Cadmium (produce)
- Manganese (produce)
- Mercury (produce and beef)
- Selenium (beef).

SEGMENT 8 - CURRENT

The sampled portion of Segment 8 includes both open and residential land uses. Summary risk estimates for exposures associated with current land use at Segment 8 are presented in Table 5.27.

Noncancer Effects

Open Land Use. The combined HIs for open land use at Segment 8 are all below the EPA target for noncancer effects.

Residential Land Use. The produce ingestion pathway exceeds the upper limit of the EPA target for noncancer effects (HI greater than 1). The HI for soil ingestion is at the limit of the EPA target (HI = 1). The remaining exposure pathway HIs are relatively minor, falling below the EPA noncancer target.

Substances with individual HQs greater than 1 for the produce ingestion pathway include arsenic, cadmium, manganese, and mercury. Other substances with HQs less than 1, but combine to yield an HI greater than 1, include copper, nickel, silver, and zinc. For the current soil ingestion pathway, only two substances represent significant percentages of the total soil ingestion HI: arsenic (12%) and mercury (76%).

Cancer Effects

Open Land Use. The combined cancer risks for open land use at Segment 8 are on the order of 10^{-6}, which is within the EPA target cancer risk range. The greatest cancer risks are related to nonradionuclide substances, and radionuclide cancer risks are all below the EPA target cancer risk range.

Nonradionuclide cancer risks for the soil ingestion and dermal exposure routes fall within the EPA target cancer risk range. Arsenic and manganese represent the bulk of the total open land use cancer risk. All remaining carcinogens and exposure routes have cancer risks of less than 10^{-6}.

Table 5.27. Risk characterization summary for EFPC Segment 8: RME estimates for current land uses

		AGRICULTURAL LAND USE						COMMERCIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		NA	NA					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	NA

		OPEN LAND USE						RESIDENTIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	2.41E−01 B	1.27E−01 B	2.80E−06 W	4.90E−06 W	1.02E−07 B	2.56E−07 B	1.06E+00 *B*	2.95E−01 B	1.23E−05 W	1.14E−05 W	2.39E−07 B	5.98E−07 B
	Dermal Contact	1.48E−02 B	1.37E−02 B	3.51E−07 B	1.09E−06 W	NA	NA	4.18E−02 B	3.20E−02 B	9.96E−07 B	2.54E−06 W	NA	NA
Surface Water	Ingestion	1.48E−03 B	1.04E−03 B	3.10E−09 B	7.22E−09 B	4.89E−10 B	1.63E−09 B	2.32E−02 B	8.62E−03 B	4.85E−08 B	6.01E−08 B	4.07E−09 B	1.36E−08 B
	Dermal Contact	1.28E−03 B	1.18E−03 B	3.01E−08 B	9.31E−08 B	NA	NA	1.73E−02 B	1.32E−02 B	4.08E−07 B	1.04E−06 W	NA	NA
Sediment	Dermal Contact	9.90E−05 B	9.19E−05 B	1.26E−08 B	3.89E−08 B	NA	NA	8.66E−04 B	6.62E−04 B	1.10E−07 B	2.80E−07 B	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	8.40E+01 *B*	4.05E+01 *B*	2.02E−04 *B*	3.25E−04 *B*	6.98E−07 B	3.02E−06 W
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	2.01E−01 B	9.63E−02 B	4.68E−05 W	7.47E−05 W	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		2.6E−01 B	1.4E−01 B					8.5E+01 *B*	4.1E+01 *B*				
Excess Lifetime Cancer Risk: Combined Exposure				3E−06 W	6E−06 W					3E−04 *B*	4E−04 *B*		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						1E−07 B	3E−07 B					9E−07 B	4E−06 W

B – below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1 x 10^{-6})
W – within EPA target cancer risk range (ELCR>1 x 10^{-6} and <1 x 10^{-4})
B – exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1 x 10^{-4})

Residential Land Use. The greatest cancer risks for the current resident scenario at Segment 8 are associated with the produce and fish ingestion pathways, but only the produce pathway exceeds EPA targets. The total nonradionuclide cancer risks for all exposures under residential land use at Segment 8 exceed the EPA target cancer risk range. Produce ingestion represents 67% and fish ingestion 16% of the total cancer risk estimate. The remaining exposure pathways combine to yield a cancer risk of 3×10^{-6}.

Two carcinogenic substances of concern for produce ingestion at Segment 8 are arsenic and beryllium. Both have individual cancer risks exceeding 1×10^{-6} and represent 98% and 1% of the total produce ingestion cancer risk, respectively. Only arsenic has a produce ingestion cancer risk greater than the EPA target range. The primary substances of carcinogenic concern for the fish ingestion pathway are Aroclor-1254 and Aroclor-1260, which represent all of the fish ingestion cancer risk.

The cancer risk estimates for the radionuclides are approximately two orders of magnitude less than the nonradionuclides. Radionuclides in produce are associated with a cancer risk estimate (for adults) that falls within the EPA target range for cancer effects. Two substances, cesium-137+D and neptunium-237+D, are associated with cancer risks on the order of 10^{-6}.

Primary Substances of Concern

- Arsenic (produce)
- Cadmium (produce)
- Manganese (produce)
- Mercury (produce).

SEGMENT 9 - CURRENT

Current land use at Segment 9 includes only the open land use designation. Summary risk estimates for this segment are presented in Table 5.28.

Noncancer Effects

Open Land Use. The combined HIs for open land use at Segment 9 are all well below the EPA noncancer effects target of 1.

Cancer Effects

Open Land Use. The combined cancer risks for open land use at Segment 9 are on the order of 10^{-6}, which is within the target cancer risk range established by EPA. Nonradionuclide

Table 5.28. Risk characterization summary for EFPC Segment 9: RME estimates for current land uses

		AGRICULTURAL LAND USE						COMMERCIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards:													
Hazard Index (HI):		NA	NA					NA	NA				
Combined Exposure													
Excess Lifetime Cancer Risk:				NA	NA					NA	NA		
Combined Exposure													
Radiological Hazards:													
Excess Lifetime Cancer Risk:						NA	NA					NA	NA
Combined Exposure													

		OPEN LAND USE						RESIDENTIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	8.92E−02 B	4.70E−02 B	2.66E−06 W	4.67E−06 W	7.22E−08 B	1.81E−07 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	5.49E−03 B	5.10E−03 B	4.03E−07 B	1.25E−06 W	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	1.48E−03 B	1.04E−03 B	3.10E−09 B	7.22E−09 B	4.89E−10 B	1.63E−09 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	1.28E−03 B	1.18E−03 B	3.01E−08 B	9.31E−08 B	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	9.90E−05 B	9.19E−05 B	1.26E−08 B	3.89E−08 B	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards:													
Hazard Index (HI):		9.8E−02 B	5.4E−02 B					NA	NA				
Combined Exposure													
Excess Lifetime Cancer Risk:				3E−06 W	6E−06 W					NA	NA		
Combined Exposure													
Radiological Hazards:													
Excess Lifetime Cancer Risk:						7E−08 B	2E−07 B					NA	NA
Combined Exposure													

B – below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1 x 10^{-6})
W – within EPA target cancer risk range (ELCR>1 x 10^{-6} and <1 x 10^{-4})
B – exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1 x 10^{-4})

risks, in particular those associated with the soil ingestion and dermal contact pathways, are dominant. Arsenic is the only substance with a cancer risk greater than 1×10^{-6}. The remaining carcinogens all have cancer risks on the order of 10^{-7} or less. Each of the radionuclide substances is associated with cancer risks that are on the order of 10^{-8} or less.

SEWER LINE BELTWAY - CURRENT

Summary risk estimates for soil exposures associated with current land use at the three areas under consideration along the SLB are presented in Tables 5.29, 5.30, and 5.31. The three areas, Emory Valley Road, Fairbanks Avenue, and Tulane Avenue, are presented and discussed together, since the risk estimates are very similar. Open land use currently predominates along the SLB. All of the noncarcinogenic and carcinogenic risk estimates under the open land use scenario were within or below EPA targets for waste site remediation under the Superfund program. The SLB is treated separately from EFPC in that exposures are not assumed to overlap between the SLB and EFPC. Exposures at the SLB are thus limited to those related to direct contact with soil. Currently, no risks related to commercial land use are considered likely within the sampled portions of the SLB.

Noncancer Effects

Open Land Use. Tables 5.29 through 5.31 indicate that the total HI scores for combined exposure across pathways for open land use at the SLB fall below the target established by EPA (i.e., HI is less than 1).

Cancer Effects

Open Land Use. The cancer risks related to the chemical carcinogens dominate over radionuclide risks and are all on the order of 10^{-6}. The radionuclide cancer risks are nearly two orders of magnitude lower (10^{-8}) than the chemical cancer risks. The combined chemical cancer risks are within the EPA target cancer risk range, and the radionuclide cancer risks are well below the risk range. The soil ingestion route dominates, and all other estimates are below the EPA target range.

Arsenic is the only substance contributing significantly to the cancer risk estimate, accounting for nearly all of the cancer risk.

5.4.4.3 Future land use conditions

Much of the future land use analysis was predicated on the assumption that land use might shift from open to residential or agricultural/homesteading. In particular, the future scenarios

Table 5.29. Risk characterization summary for Emory Valley Road: RME risk estimates for current land uses

		AGRICULTURAL LAND USE						COMMERCIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards:													
Hazard Index (HI): Combined Exposure		NA	NA					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	NA		
Radiological Hazards:													
Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	NA

		OPEN LAND USE						RESIDENTIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	1.49E−01 B	7.82E−02 B	2.55E−06 W	4.48E−06 W	3.19E−08 B	8.01E−08 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	9.10E−03 B	8.45E−03 B	1.56E−07 B	4.85E−07 B	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards:													
Hazard Index (HI): Combined Exposure		1.6E−01 B	8.7E−02 B					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				3E−06 W	5E−06 W					NA	NA		
Radiological Hazards:													
Excess Lifetime Cancer Risk: Combined Exposure						3E−08 B	8E−08 B					NA	NA

B – below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1 x 10^{-6})
W – within EPA target cancer risk range (ELCR>1 x 10^{-6} and <1 x 10^{-4})
B – exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1 x 10^{-4})

Table 5.30. Risk characterization summary for Fairbanks Avenue: RME risk estimates for current land uses

MEDIA	ROUTE	AGRICULTURAL LAND USE: NONCANCER CHILD	AGRICULTURAL LAND USE: NONCANCER ADULT	AGRICULTURAL LAND USE: CANCER CHILD	AGRICULTURAL LAND USE: CANCER ADULT	AGRICULTURAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	AGRICULTURAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT	COMMERCIAL LAND USE: NONCANCER CHILD	COMMERCIAL LAND USE: NONCANCER ADULT	COMMERCIAL LAND USE: CANCER CHILD	COMMERCIAL LAND USE: CANCER ADULT	COMMERCIAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	COMMERCIAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards:													
Hazard Index (HI): Combined Exposure		NA	NA					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	NA		
Radiological Hazards:													
Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	NA

MEDIA	ROUTE	OPEN LAND USE: NONCANCER CHILD	OPEN LAND USE: NONCANCER ADULT	OPEN LAND USE: CANCER CHILD	OPEN LAND USE: CANCER ADULT	OPEN LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	OPEN LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT	RESIDENTIAL LAND USE: NONCANCER CHILD	RESIDENTIAL LAND USE: NONCANCER ADULT	RESIDENTIAL LAND USE: CANCER CHILD	RESIDENTIAL LAND USE: CANCER ADULT	RESIDENTIAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	RESIDENTIAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT
Soil	Ingestion	1.74E-01 B	9.17E-02 B	3.29E-06 W	5.77E-06 W	2.74E-08 B	6.88E-08 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	1.07E-02 B	9.91E-03 B	2.01E-07 B	6.24E-07 B	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards:													
Hazard Index (HI): Combined Exposure		1.8E-01 B	1.0E-01 B					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				3E-06 W	6E-06 W					NA	NA		
Radiological Hazards:													
Excess Lifetime Cancer Risk: Combined Exposure						3E-08 B	7E-08 B					NA	NA

B – below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1 x 10^{-6})
W – within EPA target cancer risk range (ELCR>1 x 10^{-6} and <1 x 10^{-4})
B – exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1 x 10^{-4})

Table 5.31. Risk characterization summary for Tulane Avenue: RME risk estimates for current land uses

		AGRICULTURAL LAND USE						COMMERCIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		NA	NA					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	NA

		OPEN LAND USE						RESIDENTIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	1.52E−01 B	7.99E−02 B	3.96E−06 W	6.95E−06 W	1.86E−08 B	4.66E−08 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	9.30E−03 B	8.63E−03 B	2.43E−07 B	7.52E−07 B	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		1.6E−01 B	8.9E−02 B					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				4E−06 W	8E−06 W					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						2E−08 B	5E−08 B					NA	NA

B – below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1×10^{-6})
W – within EPA target cancer risk range (ELCR>1×10^{-6} and <1×10^{-4})
B – exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1×10^{-4})

could include home vegetable gardens and other food chain pathway exposures that may not presently be occurring. As noted in Sect. 5.2.1, residential development of property near EFPC is not unusual, so continuation of the residential trend is a reasonable possibility. Note that for the SLB, land use is unlikely to change from the current open designation.

The sampled portion of Segments 1 through 9 are currently within a 100-year floodplain, and residential development would have to be preceded by filling and grading the property to an elevation above the high water mark. The exposures developed in the BRA for future residential land use have been based on current sampling conditions, and exposures may be mitigated in the future if the contaminated soils were stabilized and capped by clean fill. Thus, the risk assessment is likely to overestimate future residential exposures. This approach is nonetheless plausible in that the fill material might be contaminated by the sampled soils during earth moving and excavation operations.

ALL SEGMENTS - GROUNDWATER - FUTURE

The following section characterizes the risk for groundwater at all nine segments of EFPC. It is important to reiterate that groundwater at EFPC is not currently used, and thus all groundwater exposures are assumed to hypothetically occur in the future. The data set for groundwater is composed of monitoring data from all nine segments at EFPC. Although the exposure point concentrations for the groundwater pathway are the same for all segments, the land uses differ.

Three of the land use scenarios (i.e., residential, agricultural/homesteader, and commercial) include groundwater exposures at some (but not all) of the segments. For the residential receptor, this includes Segments 1, 2, 3, 4, 5, 6, 8, and 9; for the agricultural/homesteader receptor, Segments 7 and 9; and for the commercial receptor, Segments 1, 2, 3, 4, and 5. The exposure assumptions for the residential and agricultural settling is the same across all segments. This includes hypothetical ingestion of drinking water and inhalation exposure to volatiles released during showering. The commercial scenario includes hypothetical use of groundwater as a drinking water source only. The groundwater risk estimates are presented in the tables for all nine segments. However, since the groundwater results are the same at all segments, they are discussed in the text only once.

Groundwater Noncancer Effects

Residential and Agricultural/Homesteader. The groundwater exposures and risk estimates for the agricultural scenario are identical to those in the residential scenario (i.e., same exposure assumptions and exposure point concentrations). RME noncancer estimates for groundwater

exposures are greater than the EPA target for noncancer effects, and are largely dominated by the ingestion route. The corresponding MLE HIs are also above the EPA target.

Commercial. The RME HI for the commercial receptor is above the EPA noncancer target, and the corresponding MLE estimate is also above the target. The only exposure route for this receptor is ingestion of groundwater.

For both of these receptors, three chemicals are responsible for the majority of the projected noncancer effects, each with HQs at or above the EPA noncancer limit. Arsenic represents 3%, mercury 9%, and manganese 85% of the groundwater ingestion HI. When combined, the remaining analytes yield to a maximum HI (for resident children) of slightly more than 1.

Groundwater Cancer Effects

Residential and Agricultural/Homesteader. As noted, groundwater exposures for the agricultural scenario are identical to those of the residential scenario. The RME cancer risk for the resident exceeds the EPA target cancer risk range, and is dominated by the ingestion route. The corresponding MLE cancer risks are on the order of 10^{-5}, which is within the EPA target cancer risk range.

Commercial. The nonradionuclide cancer risk for adults ingesting groundwater is at the limit of the EPA target cancer risk range. The radionuclide cancer risk for the commercial receptor drinking groundwater is within the EPA target cancer risk range.

Two substances represent 97% of the nonradionuclide cancer risks: arsenic with 47% and beryllium with 50% of the total cancer risk. The risks due to arsenic and beryllium slightly exceed the upper limit of the EPA target cancer risk range. The remaining nonradionuclide analyte, methylene chloride, has a cancer risk of 4×10^{-6}, which is at the lower end of the target range.

Radionuclides contribute to the total cancer risk to a lesser extent than the nonradionuclides. The greatest radionuclide cancer risks are for groundwater ingestion. These risks are within the EPA target cancer risk range, and are fairly evenly distributed between the detected radium and the uranium-234 and 238+D radionuclides. Neptunium-237 was detected in the groundwater, but was not included in the risk characterization because of large analytical counting errors reported in the sample data set. In some cases, the counting errors exceeded the upper and lower limits of the reported activity concentration. No meaningful quantification of activity for neputium in groundwater could thus be determined, and it was excluded from the quantitative risk characterization.

Primary Substances of Concern for Groundwater

- Arsenic
- Beryllium
- Manganese
- Mercury.

Comparison of Filtered and Unfiltered Groundwater Samples. The risk assessment is primarily based on unfiltered groundwater samples from monitoring wells, not fully developed drinking water supply wells. As a point of comparison, additional risk estimates are provided in this section for exposures using filtered groundwater sample results, which may better approximate conditions in a properly developed drinking water supply well.

Exposure assumptions selected for this comparison are from the agricultural land use scenario, because its receptor is the most highly exposed. The agricultural and residential exposure assumptions for groundwater are the same. Groundwater data have been aggregated across all nine segments in deriving exposure point concentrations. The conclusions presented below are therefore applicable to all other segments where groundwater exposure has been considered.

As an additional point of comparison, the UCL unfiltered concentrations of COCs in groundwater were compared with federal MCLs under SDWA. Unfiltered concentrations of mercury and cadmium exceeded MCLs. Concentrations of lead exceeded the "action level" of 15 μg/L established for concentrations at the consumer's tap. However, concentrations of the filtered samples (i.e., dissolved levels) did not exceed MCLs.

Table 5.32, briefly compares unfiltered versus filtered risk estimates for groundwater ingestion.

Table 5.32. Comparison of unfiltered versus filtered groundwater ingestion risk estimates

	Child		Adult	
	Noncancer	Cancer	Noncancer	Cancer
Unfiltered	8.4	1.5×10^{-4}	7.1	4.1×10^{-4}
Filtered	3.5	1.4×10^{-4}	3.0	4.0×10^{-4}

Filtered Versus Unfiltered Groundwater Noncancer Effects

Agricultural/Homesteader. As shown in the above table, the noncancer risk estimates decrease by more than a factor of two when using the filtered samples. UCL concentrations of mercury fell from approximately 27 μg/L in unfiltered samples to just over 1 μg/L in filtered samples. This difference of more than a factor of 20 resulted in a decrease in an HQ score for mercury (children) from nearly 3.0 to approximately 0.15.

Filtered Versus Unfiltered Groundwater Cancer Effects

Agricultural/Homesteader. The results from the cancer risk estimates are shown in Table 5.32. These risks also decreased from relatively high levels using the unfiltered samples to relatively low levels using the filtered samples. This is because the exposure point concentration for all substances in the filtered samples is lower than the unfiltered samples.

SEGMENT 1 - FUTURE

Summary risk estimates for exposures associated with future land use at Segment 1 are presented in Table 5.33.

Noncancer Effects

Residential. Under future residential land use assumptions, the noncancer HIs for soil, groundwater (discussed earlier), and produce exceed the EPA target of 1. Produce ingestion clearly dominates these estimates with HIs of 3×10^2 (children) and 1×10^2 (adults). Soil ingestion HIs are much lower, at 3 (children) and 0.9 (adults). MLE estimates for soil ingestion are all below the EPA target for noncancer effects.

Four substances dominate the produce HI estimate, each with ingestion HQs exceeding 1, representing more than 99% of the HI for produce ingestion. Mercury represents 95%, cadmium 2%, arsenic 1%, and manganese 1% of the total produce ingestion HI. For soil ingestion, only arsenic has an ingestion HQ exceeding 1, representing more than 92% of the HI for soil ingestion.

Cancer Effects

Residential. The combined exposure cancer risks for future residential land use exceed the EPA target for cancer risk. The nonradionuclide cancer risks are approximately an order of magnitude greater than the radionuclides, and only the nonradiologic components of the cancer risk exceed the EPA target.

Table 5.33. Risk characterization summary for EFPC Segment 1: RME estimates for future land use

MEDIA	ROUTE	AGRICULTURAL LAND USE: NONCANCER CHILD	AGRICULTURAL LAND USE: NONCANCER ADULT	AGRICULTURAL LAND USE: CANCER CHILD	AGRICULTURAL LAND USE: CANCER ADULT	AGRICULTURAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	AGRICULTURAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT	COMMERCIAL LAND USE: NONCANCER CHILD	COMMERCIAL LAND USE: NONCANCER ADULT	COMMERCIAL LAND USE: CANCER CHILD	COMMERCIAL LAND USE: CANCER ADULT	COMMERCIAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	COMMERCIAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	9.89E+00 *E*	NA	7.61E−05 W	NA	7.00E−06 W
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index: Combined Exposure		NA	NA					NA	9.9E+00 *E*				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	8E−05 W		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	7E−06 W

MEDIA	ROUTE	OPEN LAND USE: NONCANCER CHILD	OPEN LAND USE: NONCANCER ADULT	OPEN LAND USE: CANCER CHILD	OPEN LAND USE: CANCER ADULT	OPEN LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	OPEN LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT	RESIDENTIAL LAND USE: NONCANCER CHILD	RESIDENTIAL LAND USE: NONCANCER ADULT	RESIDENTIAL LAND USE: CANCER CHILD	RESIDENTIAL LAND USE: CANCER ADULT	RESIDENTIAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	RESIDENTIAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	3.24E+00 *E*	9.05E−01 B	2.87E−05 W	2.67E−05 W	2.70E−07 B	6.77E−07 B
	Dermal Contact	NA	NA	NA	NA	NA	NA	1.28E−01 B	9.82E−02 B	7.18E−06 W	1.83E−05 W	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	2.32E−02 B	8.62E−03 B	4.85E−08 B	6.01E−08 B	4.07E−09 B	1.36E−08 B
	Dermal Contact	NA	NA	NA	NA	NA	NA	1.73E−02 B	1.32E−02 B	4.08E−07 B	1.04E−06 W	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	8.66E−04 B	6.62E−04 B	1.10E−07 B	2.80E−07 B	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	3.28E+01 *E*	2.77E+01 *E*	9.08E−05 W	2.56E−04 *E*	3.10E−06 W	2.35E−05 W
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	7.30E−04 B	1.61E−04 B	1.33E−07 B	9.78E−08 B	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	2.79E+02 *E*	1.35E+02 *E*	1.91E−04 *E*	3.07E−04 *E*	3.26E−07 B	1.41E−06 W
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	2.01E−01 B	9.63E−02 B	4.68E−05 W	7.47E−05 W	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index: Combined Exposure		NA	NA					3.2E+02 *E*	1.6E+02 *E*				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					4E−04 *E*	7E−04 *E*		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					4E−06 W	3E−05 W

B – below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1 x 10^{-6})
W – within EPA target cancer risk range (ELCR> 1 x 10^{-6} and < 1 x 10^{-4})
E – exceeds EPA target for noncancer hazard index (HI>1), or cancer risk (ELCR< 1 x 10^{-4})

The cancer risks are primarily driven by soil, groundwater (discussed earlier), beef, and fish exposures. Soil ingestion cancer risks are all within the EPA target cancer risk range. Soil dermal contact cancer risks are lower, although they also are within the target range. The most important substances in this regard are arsenic, beryllium, Aroclor-1260, and benzo(a)pyrene. The soil ingestion cancer risks for each of these substances are greater than 1×10^{-6}.

Cancer risks for the produce pathway exceed the upper limit of the EPA target (i.e., 1×10^{-4}). Fish ingestion is associated with cancer risks that are within the target cancer risk range. The primary substance of concern for the produce ingestion pathway is arsenic, which accounts for more than 90% of the cancer risk. The remaining carcinogens combine to yield a cancer risk of 1×10^{-5}, which is within the EPA target cancer risk range.

The surface water ingestion, dermal contact, sediment dermal contact, and groundwater inhalation pathways are relatively minor. These remaining risks account for less than 2% of the total cancer risk.

Primary Substances of Concern

- Arsenic (produce)
- Cadmium (produce)
- Manganese (produce)
- Mercury (produce).

SEGMENT 2 - FUTURE

At Segment 2, the residential exposure pathways and the commercial groundwater ingestion pathway have been evaluated under future land use. Table 5.34 presents summary risk estimates for these exposures.

Noncancer Effects

Residential. Under the future residential land use assumptions, the combined noncancer HIs exceed the EPA target noncancer limit of 1. The pathways and routes of primary concern include produce ingestion and groundwater (previously discussed). Soil ingestion resulted in HIs that are below the EPA target for noncancer effects.

Four substances are associated with produce ingestion HQs exceeding 1, representing more than 97% of the HI for produce ingestion. Arsenic represents 6%, cadmium 8%, manganese 6%, and mercury 77% of the total produce ingestion HI.

Table 5.34. Risk characterization summary for EFPC Segment 2: RME estimates for future land use

MEDIA	ROUTE	AGRICULTURAL LAND USE: NONCANCER CHILD	AGRICULTURAL LAND USE: NONCANCER ADULT	AGRICULTURAL LAND USE: CANCER CHILD	AGRICULTURAL LAND USE: CANCER ADULT	AGRICULTURAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	AGRICULTURAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT	COMMERCIAL LAND USE: NONCANCER CHILD	COMMERCIAL LAND USE: NONCANCER ADULT	COMMERCIAL LAND USE: CANCER CHILD	COMMERCIAL LAND USE: CANCER ADULT	COMMERCIAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	COMMERCIAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	9.89E+00 *B*	NA	7.61E−05 W	NA	7.00E−06 W
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index: Combined Exposure		NA	NA					NA	9.9E+00 *B*				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	8E−05 W		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	7E−06 W

MEDIA	ROUTE	OPEN LAND USE: NONCANCER CHILD	OPEN LAND USE: NONCANCER ADULT	OPEN LAND USE: CANCER CHILD	OPEN LAND USE: CANCER ADULT	OPEN LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	OPEN LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT	RESIDENTIAL LAND USE: NONCANCER CHILD	RESIDENTIAL LAND USE: NONCANCER ADULT	RESIDENTIAL LAND USE: CANCER CHILD	RESIDENTIAL LAND USE: CANCER ADULT	RESIDENTIAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	RESIDENTIAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	8.58E−01 B	2.40E−01 B	2.60E−05 W	2.42E−05 W	1.81E−07 B	4.54E−07 B
	Dermal Contact	NA	NA	NA	NA	NA	NA	3.44E−02 B	2.63E−02 B	5.39E−06 W	1.37E−05 W	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	2.32E−02 B	8.62E−03 B	4.85E−08 B	6.01E−08 B	4.07E−09 B	1.36E−08 B
	Dermal Contact	NA	NA	NA	NA	NA	NA	1.73E−02 B	1.32E−02 B	4.08E−07 B	1.04E−06 W	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	8.66E−04 B	6.62E−04 B	1.10E−07 B	2.80E−07 B	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	3.28E+01 *B*	2.77E+01 *B*	9.08E−05 W	2.56E−04 *B*	3.10E−06 W	2.35E−05 W
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	7.30E−04 B	1.61E−04 B	1.33E−07 B	9.78E−08 B	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	6.29E+01 *B*	3.03E+01 *B*	2.64E−04 *B*	4.24E−04 *B*	2.24E−07 B	9.68E−07 B
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	2.01E−01 B	9.63E−02 B	4.68E−05 W	7.47E−05 W	NA	NA
Chemical Hazards: Hazard Index: Combined Exposure		NA	NA					9.7E+01 *B*	5.8E+01 *B*				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					4E−04 *B*	8E−04 *B*		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					4E−06 W	2E−05 W

B − below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1×10^{-6})
W − within EPA target cancer risk range (ELCR>1×10^{-6} and <1×10^{-4})
B − exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1×10^{-4})

Cancer Effects

Residential. The combined exposure cancer risks under future residential land use exceed the upper limit of the EPA target cancer risk range. The nonradionuclide cancer risks are nearly two orders of magnitude greater than for the radionuclides. Only the nonradiologic component of the cancer risk exceeds the EPA target cancer risk range.

The cancer risks are primarily driven by groundwater (previously discussed) and produce exposures. Soil ingestion and fish ingestion cancer risks are within the EPA target range. The soil dermal contact cancer risks are lower, but still within the EPA target range. The surface water ingestion and dermal contact, sediment dermal contact, and groundwater volatile inhalation pathways are relatively minor. These remaining risks account for less than 1% of the total cancer risk.

The primary substance of concern for the produce ingestion cancer risk is arsenic, which accounts for 97% of the cancer risk. The remaining carcinogens in the produce pathway combine to yield a cancer risk on the order of 10^{-6}, which is within the EPA cancer risk range. For soil, substances with cancer risks greater than 1×10^{-6} include arsenic with 41%, beryllium with 11%, Aroclor-1260 with 12%, benzo(a)pyrene with 19%, and dibenzo(a,h)anthracene with 5% of the summed soil ingestion cancer risk. Arsenic, Aroclor-1260, benzo(a)pyrene, and dibenzo(a,h)anthracene each have cancer risks greater than 10^{-6} for the soil dermal contact exposure route. For fish ingestion, Aroclor-1254 and Aroclor-1260 account for all of the cancer risk estimate.

Primary Substances of Concern

- Arsenic (produce)
- Cadmium (produce)
- Manganese (produce)
- Mercury (produce).

SEGMENT 3 - FUTURE

Summary risk estimates for exposures associated with future land use at Segment 3 are presented in Table 5.35.

Noncancer Effects

Residential. Under the future residential land use assumptions, the total combined noncancer HIs exceed the EPA noncancer target of 1. The pathways and routes of primary

Table 5.35. Risk characterization summary for EFPC Segment 3: RME estimates for future land use

MEDIA	ROUTE	AGRICULTURAL LAND USE: NONCANCER CHILD	AGRICULTURAL LAND USE: NONCANCER ADULT	AGRICULTURAL LAND USE: CANCER CHILD	AGRICULTURAL LAND USE: CANCER ADULT	AGRICULTURAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	AGRICULTURAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT	COMMERCIAL LAND USE: NONCANCER CHILD	COMMERCIAL LAND USE: NONCANCER ADULT	COMMERCIAL LAND USE: CANCER CHILD	COMMERCIAL LAND USE: CANCER ADULT	COMMERCIAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	COMMERCIAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	9.89E+00 *B*	NA	7.61E−05 W	NA	7.00E−06 W
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		NA	NA					NA	9.9E+00 *B*				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	8E−05 W		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	7E−06 W

MEDIA	ROUTE	OPEN LAND USE: NONCANCER CHILD	OPEN LAND USE: NONCANCER ADULT	OPEN LAND USE: CANCER CHILD	OPEN LAND USE: CANCER ADULT	OPEN LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	OPEN LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT	RESIDENTIAL LAND USE: NONCANCER CHILD	RESIDENTIAL LAND USE: NONCANCER ADULT	RESIDENTIAL LAND USE: CANCER CHILD	RESIDENTIAL LAND USE: CANCER ADULT	RESIDENTIAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	RESIDENTIAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	1.23E+00 *B*	3.44E−01 B	2.55E−05 W	2.37E−05 W	1.62E−07 B	4.06E−07 B
	Dermal Contact	NA	NA	NA	NA	NA	NA	4.92E−02 B	3.76E−02 B	5.58E−06 W	1.42E−05 W	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	2.32E−02 B	8.62E−03 B	4.85E−08 B	6.01E−08 B	4.07E−09 B	1.36E−08 B
	Dermal Contact	NA	NA	NA	NA	NA	NA	1.73E−02 B	1.32E−02 B	4.08E−07 B	1.04E−06 W	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	8.66E−04 B	6.62E−04 B	1.10E−07 B	2.80E−07 B	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	3.28E+01 *B*	2.77E+01 *B*	9.08E−05 W	2.56E−04 *B*	3.10E−06 W	2.35E−05 W
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	7.30E−04 B	1.61E−04 B	1.33E−07 B	9.78E−08 B	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	9.83E+01 *B*	4.74E+01 *B*	2.57E−04 *B*	4.13E−04 *B*	1.65E−07 B	7.13E−07 B
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	2.01E−01 B	9.63E−02 B	4.68E−05 W	7.47E−05 W	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		NA	NA					1.3E+02 *B*	7.6E+01 *B*				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					4E−04 *B*	8E−04 *B*		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					3E−06 W	2E−05 W

B − below or equal to target noncancer hazard index (HI<1), or cancer risk (ELCR<=1 x 10^{-6})
W − within EPA target cancer risk range (ELCR>1 x 10^{-6} and <1 x 10^{-4})
B − exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1 x 10^{-4})

concern include produce ingestion and groundwater ingestion (previously discussed). Soil ingestion resulted in an HI for children of 1, which is at the lower limit of the EPA target. Four substances are associated with produce ingestion HQs exceeding 1, representing approximately 99% of the HI for produce ingestion. Mercury represents approximately 87%, cadmium 3%, arsenic 4%, and manganese 5% of the total produce ingestion HI.

Cancer Effects

Residential. The combined exposure cancer risks under future residential land use exceed the EPA target for cancer risk. The nonradionuclide cancer risks were greater than those for the radionuclides, the latter of which fall within the EPA target range of 10^{-6} to 10^{-4}. Only the nonradiologic component of the cancer risk exceeds EPA target.

The cancer risks are primarily driven by soil, groundwater, and food chain exposures. The soil ingestion cancer risks are within the EPA target range, at 3×10^{-5} (children) and 2×10^{-5} (adults). The soil ingestion route dominates the dermal contact route, although risks from both are within the EPA cancer risk target range. Produce ingestion is associated with cancer risks that are higher, and exceed the the EPA target upper limit of 1×10^{-4}. Fish ingestion is associated with cancer risks that are within the target cancer risk range. The risks from the residential surface water, sediment, and groundwater volatile inhalation pathways are relatively minor, accounting for less than 1% of the total cancer risk.

The primary substance of concern for the produce ingestion cancer risk is arsenic, which accounts for 96% of the cancer risk. Produce ingestion cancer risks greater than 1×10^{-6} also are associated with beryllium and benzo(a)pyrene, although these and all of the remaining carcinogens combine to a cancer risk on the order of 10^{-6}, which is within the EPA target cancer risk range.

Primary Substances of Concern

- Arsenic (produce)
- Cadmium (produce)
- Manganese (produce)
- Mercury (produce).

SEGMENT 4 - FUTURE

The future summary risk estimates for exposures associated with land use at Segment 4 are shown in Table 5.36.

Table 5.36. Risk characterization summary for EFPC Segment 4: RME estimates for the future land uses

MEDIA	ROUTE	AGRICULTURAL LAND USE: NONCANCER CHILD	AGRICULTURAL LAND USE: NONCANCER ADULT	AGRICULTURAL LAND USE: CANCER CHILD	AGRICULTURAL LAND USE: CANCER ADULT	AGRICULTURAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	AGRICULTURAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT	COMMERCIAL LAND USE: NONCANCER CHILD	COMMERCIAL LAND USE: NONCANCER ADULT	COMMERCIAL LAND USE: CANCER CHILD	COMMERCIAL LAND USE: CANCER ADULT	COMMERCIAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	COMMERCIAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	9.89E+00 *B*	NA	7.61E−05 W	NA	7.00E−06 W
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		NA	NA					NA	9.9E+00 *B*				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	8E−05 W		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	7E−06 W

MEDIA	ROUTE	OPEN LAND USE: NONCANCER CHILD	OPEN LAND USE: NONCANCER ADULT	OPEN LAND USE: CANCER CHILD	OPEN LAND USE: CANCER ADULT	OPEN LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	OPEN LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT	RESIDENTIAL LAND USE: NONCANCER CHILD	RESIDENTIAL LAND USE: NONCANCER ADULT	RESIDENTIAL LAND USE: CANCER CHILD	RESIDENTIAL LAND USE: CANCER ADULT	RESIDENTIAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	RESIDENTIAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	3.60E+00 *B*	1.00E+00 *B*	3.96E−05 W	3.68E−05 W	1.96E−07 B	4.91E−07 B
	Dermal Contact	NA	NA	NA	NA	NA	NA	1.42E−01 B	1.09E−01 B	9.94E−06 W	2.53E−05 W	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	2.32E−02 B	8.62E−03 B	4.85E−08 B	6.01E−08 B	4.07E−09 B	1.36E−08 B
	Dermal Contact	NA	NA	NA	NA	NA	NA	1.73E−02 B	1.32E−02 B	4.08E−07 B	1.04E−06 W	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	8.66E−04 B	6.62E−04 B	1.10E−07 B	2.80E−07 B	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	328E+01 *B*	2.77E+01 *B*	9.08E−05 W	2.56E−04 *B*	3.10E−06 W	2.35E−05 W
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	7.30E−04 B	1.61E−04 B	1.33E−07 B	9.78E−08 B	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	3.07E+02 *B*	1.48E+02 *B*	2.95E−04 *B*	4.74E−04 *B*	3.65E−07 B	1.58E−06 W
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	2.01E−01 B	9.63E−02 B	4.68E−05 W	7.47E−05 W	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		NA	NA					3.4E+02 *B*	1.8E+02 *B*				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					5E−04 *B*	9E−04 *B*		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					4E−06 W	3E−05 W

B − below or equal to limit of noncancer hazard index (HI<=1), or cancer risk (ELCR<=1 x 10^{-6})
W − within EPA target cancer risk range (ELCR>1 x 10^{-6} and <1 x 10^{-4})
B − exceeds EPA target for noncancer hazard index (HI>1), or cancer risk (ELCR>1 x 10^{-4})

Noncancer Effects

Residential. Under the future residential land use assumptions, EPA targets for noncancer risks are exceeded (HIs greater than 1). Produce ingestion is the primary pathway and route of concern, although the HI for soil ingestion also exceeds the EPA target. The substances responsible for the majority of the noncancer risk estimate for the produce pathway are arsenic, cadmium, manganese, and mercury. Each substance has an HQ at or exceeding 1. For soil ingestion, mercury is the only substance with an HI greater than 1, and only for children.

Cancer Effects

Residential. The cancer risks for future residential land use exceed the upper limit of the EPA target cancer risk range. The elevated risks are associated with exposure to chemical carcinogens. Risks associated with exposure to radionuclides fall within the target ranges established by EPA.

The cancer risks are primarily driven by groundwater ingestion (discussed previously) and produce ingestion. Substances with produce ingestion cancer risks exceeding 1×10^{-6} include arsenic (with a cancer risk greater than 1×10^{-4}), beryllium, Aroclor-1254, Aroclor-1260, and benzo(a)pyrene.

The soil dermal contact and fish ingestion pathways are each associated with cancer risks that fall within the EPA target cancer risk range. For soil ingestion, substances with cancer risks exceeding 1×10^{-6} include arsenic, beryllium, Aroclor-1254, Aroclor-1260, and benzo(a)pyrene. The remaining exposures (surface water ingestion, dermal contact, sediment dermal contact, and groundwater inhalation) all fall below the EPA target cancer risk range.

Primary Substances of Concern

- Arsenic (produce)
- Cadmium (produce)
- Manganese (produce)
- Mercury (soil and produce).

SEGMENT 5 - FUTURE

Summary risk estimates for exposures associated with future land use at Segment 5 are presented in Table 5.37.

Table 5.37. Risk characterization summary for EFPC Segment 5: RME risk estimates for future land uses

		AGRICULTURAL LAND USE						COMMERCIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	9.89E+00 *B*	NA	7.61E−05 W	NA	7.00E−06 W
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards:													
Hazard Index (HI): Combined Exposure		NA	NA					NA	9.9E+00 *B*				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	8E−05 W		
Radiological Hazards:													
Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	7E−06 W

		OPEN LAND USE						RESIDENTIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	1.23E+00 *B*	3.45E−01 B	1.99E−05 W	1.85E−05 W	1.17E−07 B	2.93E−07 B
	Dermal Contact	NA	NA	NA	NA	NA	NA	4.92E−02 B	3.76E−02 B	4.15E−06 W	1.06E−05 W	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	2.32E−02 B	8.62E−03 B	4.85E−08 B	6.01E−08 B	4.07E−09 B	1.36E−08 B
	Dermal Contact	NA	NA	NA	NA	NA	NA	1.73E−02 B	1.32E−02 B	4.08E−07 B	1.04E−06 W	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	8.66E−04 B	6.62E−04 B	1.10E−07 B	2.80E−07 B	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	3.28E+01 *B*	2.77E+01 *B*	9.08E−05 W	2.36E−04 *B*	3.10E−06 W	2.35E−05 W
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	7.30E−04 B	1.61E−04 B	1.33E−07 B	9.78E−08 B	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	9.87E+01 *B*	4.76E+01 *B*	2.50E−04 *B*	4.02E−04 *B*	2.28E−07 B	9.85E−07 B
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	2.01E−01 B	9.63E−02 B	4.68E−05 W	7.47E−05 W	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards:													
Hazard Index (HI): Combined Exposure		NA	NA					1.3E+02 *B*	7.6E+01 *B*				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					4E−04 *B*	8E−04 *B*		
Radiological Hazards:													
Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					3E−06 W	2E−05 W

B − below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1×10^{-6})
W − within EPA target cancer risk range (ELCR>1×10^{-6} and <1×10^{-4})
B − exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1×10^{-4})

Noncancer Effects

Residential. Under the future residential land use assumptions, the total combined noncancer HI exceeds the target of 1 established by EPA. The pathways and routes of primary concern are produce and groundwater ingestion (which was previously discussed). The HIs for these pathways exceed 1. The HI for the soil ingestion pathway for children is at the limit (HI = 1).

Four substances are associated with produce ingestion HQs exceeding 1, and represent approximately 98% of the produce ingestion HI. Mercury is associated with approximately 85%, cadmium 5%, arsenic 4%, and manganese 5% of the total produce ingestion HI.

Cancer Effects

Residential. The combined exposure cancer risks under future residential land use exceed the EPA target cancer risk range. The greatest combined radionuclide cancer risk is on the order of 10^{-5}, which is within the EPA target range.

The cancer risks are primarily driven by soil, groundwater, and food chain exposures. Only the produce ingestion and groundwater ingestion (discussed earlier) exceed the EPA target upper limit of 1×10^{-4}. The soil ingestion, dermal contact, and fish ingestion cancer risks each exceed 1×10^{-6}, but are within the EPA target cancer risk range.

The residential surface water ingestion, dermal contact, sediment dermal contact, and groundwater volatile inhalation pathways are relatively minor. These remaining risks account for less than 2% of the total cancer risk, combining to yield a cancer risk of 2×10^{-6}. This is at the low end of the EPA target cancer risk range, and is mostly due to surface water dermal contact, with several organic substances that each have independent cancer risks in the 10^{-8} to 10^{-7} range.

The primary substance of concern for the produce ingestion cancer risk is arsenic, which accounts for 98% of the cancer risk. The cancer risk for the remaining carcinogens is 7×10^{-6}, which is within the EPA target cancer risk range.

Primary Substances of Concern

- Arsenic (produce)
- Cadmium (produce)
- Manganese (produce)
- Mercury (produce).

SEGMENT 6 - FUTURE

At Segment 6, the residential and agricultural/homesteader exposure pathways have been evaluated under future land use. Table 5.38 presents summary risk estimates for these exposures.

Noncancer Effects

Residential. Under the future residential land use assumptions, the total combined noncancer HIs exceed the EPA target of 1. The pathways and routes of primary concern include produce and groundwater ingestion (discussed earlier). The HIs for these pathways exceed 1. Soil ingestion HIs are below the EPA target.

Four substances are associated with produce ingestion HQs exceeding 1, representing approximately 99% of the produce ingestion HI. Arsenic represents about 6%, cadmium 4%, manganese 10%, and mercury 78% of the total produce ingestion HI.

Cancer Effects

Residential. The combined cancer risks for chemical contaminants exceed the EPA target cancer risk range. Risks associated with exposure to radionuclides fall within the target range.

The cancer risks are primarily driven by groundwater (discussed earlier) and produce ingestion exposures. Produce ingestion cancer risks exceed the upper limit of the EPA target range (1×10^{-4}). The soil and fish ingestion cancer risks are less and fall within the EPA target range.

The residential surface water, sediment, and groundwater volatile inhalation pathways are relatively minor. These remaining risks account for less than 2% of the total cancer risk, combining to yield a cancer risk of 2×10^{-6}. This is at the low end of the EPA target cancer risk range, and is mostly due to surface water dermal contact, with several organic substances that each have independent cancer risks in the 10^{-8} to 10^{-7} range.

The primary substance of concern for the produce ingestion cancer risk is arsenic, which accounts for 98% of the cancer risk. The remaining carcinogens are responsible for a cancer risk of 6×10^{-6}, which is within the EPA target cancer risk range.

The combined radionuclide cancer risks are within the EPA target cancer risk range, and the risks are evenly distributed between radium-228+D, uranium-234, and uranium-238+D.

Table 5.38. Risk characterization summary for EFPC Segment 6: RME estimates for future land uses

MEDIA	ROUTE	AGRICULTURAL LAND USE						COMMERCIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
		CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		NA	NA					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	NA

MEDIA	ROUTE	OPEN LAND USE						RESIDENTIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
		CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	6.69E−01 B	1.87E−01 B	1.32E−05 W	1.23E−05 W	2.30E−07 B	5.76E−07 B
	Dermal Contact	NA	NA	NA	NA	NA	NA	2.65E−02 B	2.03E−02 B	1.65E−06 W	4.21E−06 W	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	2.32E−02 B	8.62E−03 B	4.85E−08 B	6.01E−08 B	4.07E−09 B	1.36E−08 B
	Dermal Contact	NA	NA	NA	NA	NA	NA	1.73E−02 B	1.32E−02 B	4.08E−07 B	1.04E−06 W	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	8.66E−04 B	6.62E−04 B	1.10E−07 B	2.80E−07 B	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	3.28E+01 *B*	2.77E+01 *B*	9.08E−05 W	2.56E−04 *B*	3.10E−06 W	2.35E−05 W
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	7.30E−04 B	1.61E−04 B	1.33E−07 B	9.78E−08 B	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	5.05E+01 *B*	2.44E+01 *B*	1.97E−04 *B*	3.16E−04 *B*	1.76E−07 B	7.60E−07 B
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	2.01E−01 B	9.63E−02 B	4.68E−05 W	7.47E−05 W	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		NA	NA					8.4E+01 *B*	5.2E+01 *B*				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					3E−04 *B*	7E−04 *B*		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					4E−06 W	2E−05 W

B – below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1 x 10^{-6})
W – within EPA target cancer risk range (ELCR>1 x 10^{-6} and <1 x 10^{-4})
B – exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1 x 10^{-4})

Primary Substances of Concern

- Arsenic (produce)
- Cadmium (produce)
- Manganese (produce)
- Mercury (produce).

SEGMENT 7 - FUTURE

The risk evaluation at Segment 7 was conducted for the agricultural and open land use exposure pathways. Summary risk estimates for these exposures are presented in Table 5.39.

Noncancer Effects

Open Land Use. Under the future open land use assumptions, the total combined noncancer HIs are well below the EPA target of 1, at 0.1 (children) and 0.07 (adults).

Agricultural Land Use. Under the future agricultural land use assumptions, the total combined noncancer HIs exceed the EPA target. The pathways and routes of primary concern each have HIs greater than 1, and include ingestion of produce, beef, and dairy products. Soil ingestion HIs are below the EPA noncancer effects target.

Four substances are associated with produce ingestion HQs exceeding 1, representing approximately 98% of the produce ingestion HI. Mercury represents 59%, cadmium 10%, arsenic 8%, and manganese 20% of the total produce ingestion HI.

For beef ingestion, four substances have HQs exceeding 1, representing nearly 95% of the total HI. Arsenic represents 6%, mercury 68%, selenium 6%, and zinc 13% of the total beef ingestion HI.

For dairy product ingestion, eight substances are associated with HQs at or exceeding the EPA target of 1. Barium represents 3%, cadmium 5%, copper 8%, manganese 10%, mercury 45%, selenium 8%, silver 3%, and zinc 18% of the total HI for milk ingestion.

Harvesting of Hay. The noncancer HI for soil dust inhalation due to future agricultural mowing activities is 0.2, which is below the EPA target of 1 for noncancer effects.

Table 5.39. Risk characterization summary for EFPC Segment 7: RME risk estimates for future land uses

		AGRICULTURAL LAND USE						COMMERCIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	5.34E−01 B	1.49E−01 B	1.41E−05 W	1.32E−05 W	2.17E−07 B	5.43E−07 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	2.12E−02 B	1.62E−02 B	1.64E−06 W	4.19E−06 W	NA	NA	NA	NA	NA	NA	NA	NA.
Surface Water	Ingestion	2.32E−02 B	8.62E−03 B	4.85E−08 B	6.01E−08 B	4.07E−09 B	1.36E−08 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	1.73E−02 B	1.32E−02 B	4.08E−07 B	1.04E−06 W	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	8.66E−04 B	6.62E−04 B	1.10E−07 B	2.80E−07 B	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	3.28E+01 *B*	2.77E+01 *B*	9.08E−05 W	2.56E−04 *B*	3.10E−06 W	2.35E−05 W	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	7.30E−04 B	1.61E−04 B	1.33E−07 B	9.78E−08 B	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	9.04E+01 *B*	4.36E+01 *B*	4.92E−04 *B*	7.91E−04 *B*	7.16E−07 B	3.10E−06 W	NA	NA	NA	NA	NA	NA
Beef	Ingestion	2.01E+01 *B*	1.01E+01 *B*	1.66E−04 *B*	2.79E−04 *B*	2.21E−06 W	9.99E−06 W	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	3.68E+01 *B*	8.89E+00 *B*	2.48E−04 *B*	2.00E−04 *B*	3.46E−06 W	7.51E−06 W	NA	NA	NA	NA	NA	NA
Fish	Ingestion	2.01E−01 B	9.63E−02 B	4.68E−05 W	7.47E−05 W	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	2.34E−01	NA	2.55E−06	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		1.8E+02 *B*	9.1E+01 *B*					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				1E−03 *B*	2E−03 *B*					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						1E−05 W	4E−05 W					NA	NA

		OPEN LAND USE						RESIDENTIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	1.21E−01 B	6.39E−02 B	3.21E−06 W	5.64E−06 W	4.93E−08 B	2.33E−07 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	7.49E−03 B	6.96E−03 B	5.80E−07 B	1.80E−06 W	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	1.48E−03 B	1.04E−03 B	3.10E−09 B	7.22E−09 B	4.89E−10 B	1.63E−09 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	1.28E−03 B	1.18E−03 B	3.01E−08 B	9.31E−08 B	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	9.90E−05 B	9.19E−05 B	1.26E−08 B	3.89E−08 B	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		1.3E−01 B	7.3E−02 B					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				4E−06 W	8E−06 W					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						5E−08 B	2E−07 B					NA	NA

B – below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1 x 10^{-6})
W – within EPA target cancer risk range (ELCR>1 x 10^{-6} and <1 x 10^{-4})
B – exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1 x 10^{-4})

Cancer Effects

Open Land Use. The combined cancer risks under future open land use are within the noted EPA target range for cancer risk, the bulk of which is related to soil ingestion. The HIs for soil dermal contact, surface water, and sediment are all well below the target.

Agricultural Land Use. The combined cancer risks for nonradionuclides under future agricultural land use exceed the EPA cancer risk target. Summed nonradionuclide cancer risks are within the target range and are approximately two orders of magnitude less than those of the radionuclides.

The future agricultural cancer risks are primarily driven by soil, groundwater, and food chain exposures. The soil ingestion and dermal contact cancer risks are both within the EPA target range. Cancer risks from the produce, beef, and dairy pathways are all similar in magnitude, and all exceed the upper limit of the EPA cancer risk target. Cancer risks from fish ingestion are within the target range.

The agricultural surface water and sediment pathways are relatively minor, falling below the EPA target for all routes except dermal contact with surface water, which is at the lower limit of the target range (1×10^{-6}). This is due to dermal contact with several organic substances each with independent cancer risks in the 10^{-8} to 10^{-7} range.

The primary substance of concern for the produce ingestion cancer risk is arsenic, which accounts for 98% of the cancer risk. Only arsenic has a cancer risk that is above the upper limit of the EPA target. Other nonradionuclide carcinogens with produce ingestion cancer risks exceeding 1×10^{-6} include beryllium and benzo(a)pyrene. Radionuclide cancer risks are above 1×10^{-6} for cesium-137+D and neptunium-237+D.

For the beef pathway, only the cancer risk related to arsenic exceeded the upper limit of the EPA cancer risk target. Other nonradionuclide substances falling within the target range of 10^{-6} to 10^{-4} include beryllium, Aroclor-1260, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, and indeno(1,2,3-cd)pyrene. Three radionuclides (neptunium-237+D, thorium-228+D. and thorium-232) also are associated with beef ingestion cancer risks falling within the EPA cancer risk target range.

For the dairy pathway, none of the cancer risk estimates exceed the EPA target range, but eight nonradionuclide and two radionuclide substances fall within the target range. For the

nonradionuclides, these include arsenic, Aroclor-1260, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, and indeno(1,2,3-cd)pyrene.

Harvesting of Hay. The combined exposure cancer risk due to soil inhalation during future mowing activities at Segment 7 is within the EPA target cancer risk range at 3×10^{-6}. Arsenic is responsible for 90% of this risk.

Primary Substances of Concern

- Arsenic (produce and beef)
- Barium (milk)
- Cadmium (produce and milk)
- Manganese (produce and milk)
- Mercury (produce, beef and milk)
- Selenium (beef and milk)
- Silver (milk)
- Zinc (beef and milk).

SEGMENT 8 - FUTURE

Residential is the only land use designation considered for the future at Segment 8. Summary risk estimates for exposures associated with future land use at Segment 8 are presented in Table 5.40.

Noncancer Effects

Residential Land Use. Under the future residential land use assumptions, the total combined noncancer HIs at Segment 8 exceed the EPA target cancer range. The pathway and route of primary concern is produce ingestion, with an HI greater than 1. The soil ingestion cancer risk is at the limit of the target range (HI = 1), but only for children.

Four substances are associated with produce ingestion HQs exceeding 1, representing nearly all of the produce ingestion HI. Mercury represents about 84%, cadmium 5%, arsenic 4%, and manganese 5% of the total produce ingestion HI.

Cancer Effects

Residential land use. Only the nonradiologic component of the produce ingestion cancer risk exceeds the EPA target cancer risk range. The soil ingestion and dermal contact cancer risks are within the EPA cancer risk target range. The surface water ingestion, dermal contact, and

Table 5.40. Risk characterization summary for EFPC Segment 8: RME estimates for future land uses

		AGRICULTURAL LAND USE						COMMERCIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards:													
Hazard Index (HI): Combined Exposure		NA	NA					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	NA		
Radiological Hazards:													
Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	NA

		OPEN LAND USE						RESIDENTIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	1.06E+00 *B*	2.95E-01 B	1.23E-05 W	1.14E-05 W	2.39E-07 B	5.98E-07 B
	Dermal Contact	NA	NA	NA	NA	NA	NA	4.18E-02 B	3.20E-02 B	9.96E-07 B	2.54E-06 W	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	2.32E-02 B	8.62E-03 B	4.85E-08 B	6.01E-08 B	4.07E-09 B	1.36E-08 B
	Dermal Contact	NA	NA	NA	NA	NA	NA	1.73E-02 B	1.32E-02 B	4.08E-07 B	1.04E-06 W	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	8.66E-04 B	6.62E-04 B	1.10E-07 B	2.80E-07 B	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	3.28E+01 *B*	2.77E+01 *B*	9.08E-05 W	2.56E-04 *B*	3.10E-06 W	2.35E-05 W
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	7.30E-04 B	1.61E-04 B	1.33E-07 B	9.78E-08 B	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	8.40E+01 *B*	4.05E+01 *B*	2.02E-04 *B*	3.25E-04 *B*	6.98E-07 B	3.02E-06 W
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	2.01E-01 B	9.63E-02 B	4.68E-05 W	7.47E-05 W	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards:													
Hazard Index (HI): Combined Exposure		NA	NA					1.2E+02 *B*	6.9E+01 *B*				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					4E-04 *B*	7E-04 *B*		
Radiological Hazards:													
Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					4E-06 W	3E-05 W

B – below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1×10^{-6})
W – within EPA target cancer risk range (ELCR>1×10^{-6} and <1×10^{-4})
B – exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1×10^{-4})

sediment dermal contact pathways are relatively minor. These remaining risks account for less than 2% of the total cancer risk, and combine to yield a cancer risk that is at the low end of the EPA target cancer risk range. These minor risks are mostly due to surface water dermal contact, with several organic substances that each had independent cancer risks in the 10^{-8} to 10^{-7} range.

The primary substance of concern for the produce ingestion cancer risk is arsenic, which accounts for 98% of the cancer risk. The remaining carcinogens are associated with a cancer risk that is on the order of 10^{-6}, which is within the EPA target range.

Primary Substances of Concern

- Arsenic (produce)
- Cadmium (produce)
- Manganese (produce)
- Mercury (produce).

SEGMENT 9 - FUTURE

Both agricultural and residential land use are considered in the future scenarios for Segment 9. Summary risk estimates for exposures associated with future land use at Segment 9 are presented in Table 5.41.

Noncancer Effects

Residential Land Use. Under the future residential land use assumptions, the total combined noncancer HIs at Segment 9 exceed the EPA noncancer effects target of 1. The pathways and routes of primary concern are those with HIs greater than 1, and include only produce ingestion. Soil ingestion is related to HIs that fall below the EPA target.

Four substances are associated with individual produce ingestion HQs exceeding 1, and represent nearly all of the total produce ingestion HI. Arsenic is associated with 10%, cadmium 7%, manganese 15%, and mercury 66% of the total produce ingestion HI.

Agricultural Land Use. Under the future agricultural land use assumptions, combined HIs exceed the EPA target of 1. The pathways and routes of primary concern are those with HIs exceeding 1, and include ingestion of produce, beef, and dairy products (groundwater risks were discussed earlier). Soil ingestion is related to HIs that are below EPA noncancer targets.

Table 5.41. Risk characterization summary for East Fork Poplar Creek Segment 9: RME estimates for future land uses

MEDIA	ROUTE	AGRICULTURAL LAND USE: NONCANCER CHILD	AGRICULTURAL LAND USE: NONCANCER ADULT	AGRICULTURAL LAND USE: CANCER CHILD	AGRICULTURAL LAND USE: CANCER ADULT	AGRICULTURAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	AGRICULTURAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT	COMMERCIAL LAND USE: NONCANCER CHILD	COMMERCIAL LAND USE: NONCANCER ADULT	COMMERCIAL LAND USE: CANCER CHILD	COMMERCIAL LAND USE: CANCER ADULT	COMMERCIAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	COMMERCIAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT
Soil	Ingestion	3.92E−01 B	1.10E−01 B	1.17E−05 W	1.09E−05 W	1.68E−07 B	4.22E−07 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	1.56E−02 B	1.19E−02 B	1.14E−06 W	2.91E−06 W	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	2.32E−02 B	8.62E−03 B	4.85E−08 B	6.01E−08 B	4.07E−09 B	1.36E−08 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	1.73E−02 B	1.32E−02 B	4.08E−07 B	1.04E−06 W	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	8.66E−04 B	6.62E−04 B	1.10E−07 B	2.80E−07 B	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	3.28E+01 *E*	2.77E+01 *E*	9.08E−05 W	2.56E−04 *E*	3.10E−06 W	2.35E−05 W	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	7.30E−04 B	1.61E−04 B	1.33E−07 B	9.78E−08 B	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	6.30E+01 *E*	3.04E+01 *E*	4.29E−04 *E*	6.90E−04 *E*	1.07E−06 W	4.61E−06 W	NA	NA	NA	NA	NA	NA
Beef	Ingestion	1.77E+01 *E*	8.94E+00 *E*	1.30E−04 *E*	2.18E−04 *E*	2.06E−06 W	9.30E−06 W	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	3.06E+01 *E*	7.42E+00 *E*	1.70E−04 *E*	1.37E−04 *E*	3.44E−06 W	7.47E−06 W	NA	NA	NA	NA	NA	NA
Fish	Ingestion	2.01E−01 B	9.63E−02 B	4.68E−05 W	7.47E−05 W	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	1.30E−01	NA	2.14E−06	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		1.4E+02 *E*	7.5E+01 *E*					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				9E−04 *E*	1E−03 *E*					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						1E−05 W	5E−05 W					NA	NA

MEDIA	ROUTE	OPEN LAND USE: NONCANCER CHILD	OPEN LAND USE: NONCANCER ADULT	OPEN LAND USE: CANCER CHILD	OPEN LAND USE: CANCER ADULT	OPEN LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	OPEN LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT	RESIDENTIAL LAND USE: NONCANCER CHILD	RESIDENTIAL LAND USE: NONCANCER ADULT	RESIDENTIAL LAND USE: CANCER CHILD	RESIDENTIAL LAND USE: CANCER ADULT	RESIDENTIAL LAND USE: RADIONUCLIDES (CANCER ONLY) CHILD	RESIDENTIAL LAND USE: RADIONUCLIDES (CANCER ONLY) ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	3.92E−01 B	1.10E−01 B	1.17E−05 W	1.09E−05 W	1.68E−07 B	4.22E−07 B
	Dermal Contact	NA	NA	NA	NA	NA	NA	1.56E−02 B	1.19E−02 B	1.14E−06 W	2.91E−06 W	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	2.32E−02 B	8.62E−03 B	4.85E−08 B	6.01E−08 B	4.07E−09 B	1.36E−08 B
	Dermal Contact	NA	NA	NA	NA	NA	NA	1.73E−02 B	1.32E−02 B	4.08E−07 B	1.04E−06 W	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	8.66E−04 B	6.62E−04 B	1.10E−07 B	2.80E−07 B	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	3.28E+01 *E*	2.77E+01 *E*	9.08E−05 W	2.56E−04 *E*	3.10E−06 W	2.35E−05 W
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	7.30E−04 B	1.61E−04 B	1.33E−07 B	9.78E−08 B	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	2.70E+01 *E*	1.30E+01 *E*	1.84E−04 *E*	2.96E−04 *E*	4.57E−07 B	1.98E−06 W
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	2.01E−01 B	9.63E−02 B	4.68E−05 W	7.47E−05 W	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		NA	NA					6.0E+01 *E*	4.1E+01 *E*				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					3E−04 *E*	6E−04 *E*		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					4E−06 W	3E−05 W

B – below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1 x 10^{-6})
W – within EPA target cancer risk range (ELCR>1 x 10^{-6} and <1 x 10^{-4})
E – exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1 x 10^{-4})

Four substances are associated with produce ingestion HQs exceeding 1, and represent essentially all of the produce ingestion HI. Mercury represents approximately 66%, cadmium 7%, arsenic 10%, and manganese 15% of the total produce ingestion HI.

Two substances have beef ingestion HQs exceeding 1, representing nearly 85% of the beef ingestion HI. Mercury represents about 72% and zinc 13% of the total beef ingestion HI.

For dairy product (milk) ingestion, two substances have individual ingestion HQs exceeding 1, representing almost 70% of the total dairy ingestion HI. Mercury represents 50% and zinc 19% of the total dairy ingestion HI.

Cancer Effects

Residential Land Use. The combined cancer risks under this scenario exceed the EPA target cancer risk range, and are related to nonradionuclide substances. Radionuclide cancer risks fell within the EPA target range of 10^{-6} to 10^{-4}.

The cancer risks are primarily driven by soil and food chain exposures. The soil ingestion cancer risk is on the order of 10^{-5}, which is within the EPA target range. The soil dermal contact cancer risks are lower, and also within the EPA target range for cancer effects. Produce ingestion is associated with cancer risks that exceed the upper limit (1×10^{-4}) of the EPA cancer risk target. Fish ingestion is associated with cancer risks that are within the EPA target cancer risk range. The surface water, sediment, and groundwater volatile inhalation pathways for residential land use are relatively minor. These remaining risks account for a very small (less than 1%) percentage of the total cancer risk, and are below the EPA target cancer risk range.

The primary substance of concern for the produce ingestion cancer risk is arsenic, which accounts for 98% of the cancer risk. The remaining carcinogens sum to a cancer risk that is within the EPA target cancer risk range. The combined radionuclide cancer risks are nearly all due to groundwater ingestion (discussed earlier), and are within the EPA target cancer risk range.

Agricultural Land Use. The combined cancer risks under future agricultural land use are greater than the upper limit of the EPA cancer risk target. The nonradionuclide cancer risks are nearly two orders of magnitude greater than those of the radionuclides. Only the nonradiologic component of the cancer risk exceeds the EPA target for cancer risk, while the radionuclide cancer risks fall within the EPA target. The cancer risks are primarily driven by food chain pathway exposures, which exceed EPA cancer risk targets. The soil, surface water, sediment, and air pathways are within the EPA target cancer risk range.

Cancer risks from the produce, beef, and dairy ingestion pathways each exceed the upper limit of the EPA target of 1×10^{-4}. Cancer risks from fish ingestion fall within the EPA target range. For produce ingestion, only arsenic is associated with a cancer risk greater than 1×10^{-4}, and accounts for 98% of the total cancer risk estimate. Only one other nonradionuclide substance (beryllium) is associated with a cancer risk greater than 1×10^{-6}. For the radionuclides, cancer risks greater than 1×10^{-6} were determined for cesium-137+D and neptunium-237+D.

For beef ingestion, only arsenic is associated with a cancer risk greater than 1×10^{-4}. Beryllium, Aroclor-1260, Aroclor-1254, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, and indeno(1,2,3-cd)pyrene are each associated with cancer risks that fall within the EPA target range for cancer risks.

For dairy product (milk) ingestion, none of the carcinogens individually relate to cancer risks exceeding 1×10^{-4}. The total cancer risk estimate (which exceeds 1×10^{-4}) is due to the combination of several carcinogens. Arsenic, Aroclor-1260, Aroclor-1254, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, and indeno(1,2,3-cd)pyrene are each related to cancer risks falling within the EPA target cancer risk range.

Primary Substances of Concern

- Arsenic (produce, beef, and dairy)
- Cadmium (produce and dairy)
- Manganese (produce and dairy)
- Mercury (produce, beef, and dairy)
- Zinc (beef and dairy).

SEWER LINE BELTWAY - FUTURE

The summary risk estimates for open land use in the future at the three areas considered along the SLB are presented in Tables 5.42, 5.43, and 5.44. These three areas are presented and discussed together, since there is virtually no difference between them. Open land use currently predominates along the SLB, and little or no change is assumed over time. The risk estimates and the following discussion are thus the same as for the current land use of the SLB. All of the noncarcinogenic or carcinogenic risk estimates for the SLB fall within or below EPA targets for waste site remediation under the Superfund program. No risks related to commercial land use are likely within the sampled portions of the SLB.

Table 5.42. Risk characterization summary for Emory Valley Road: RME risk estimates for future land uses

		AGRICULTURAL LAND USE						COMMERCIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		NA	NA					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	NA

		OPEN LAND USE						RESIDENTIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	1.5E−01 B	7.8E−02 B	2.6E−06 W	4.5E−06 W	1.7E−08 B	8.0E−08 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	9.1E−03 B	8.4E−03 B	1.6E−07 B	4.8E−07 B	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		1.6E−01 B	8.7E−02 B					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				3E−06 W	5E−06 W					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						2E−08 B	8E−08 B					NA	NA

B – below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1×10^{-6})
W – within EPA target cancer risk range (ELCR>1×10^{-6} and <1×10^{-4})
B – exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1×10^{-4})

Table 5.43. Risk characterization summary for Fairbanks Avenue: RME risk estimates for future land uses

MEDIA	ROUTE	AGRICULTURAL LAND USE						COMMERCIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
		CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards:													
Hazard Index (HI): Combined Exposure		NA	NA					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	NA		
Radiological Hazards:													
Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	NA

MEDIA	ROUTE	OPEN LAND USE						RESIDENTIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
		CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	1.7E-01 B	9.2E-02 B	3.3E-06 W	5.8E-06 W	1.5E-08 B	6.9E-08 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	1.1E-02 B	9.9E-03 B	2.0E-07 B	6.2E-07 B	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards:													
Hazard Index (HI): Combined Exposure		1.8E-01 B	1.0E-01 B					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				3E-06 W	6E-06 W					NA	NA		
Radiological Hazards:													
Excess Lifetime Cancer Risk: Combined Exposure						1E-08 B	7E-08 B					NA	NA

B – below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1×10^{-6})
W – within EPA target cancer risk range (ELCR>1×10^{-6} and <1×10^{-4})
B – exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1×10^{-4})

Table 5.44. Risk characterization summary for Tulane Avenue: RME risk estimates for future land uses

		AGRICULTURAL LAND USE						COMMERCIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		NA	NA					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	NA

		OPEN LAND USE						RESIDENTIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	1.5E−01 B	8.0E−02 B	4.0E−06 W	7.0E−06 W	9.9E−09 B	4.7E−08 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	9.3E−03 B	8.6E−03 B	2.4E−07 B	7.5E−07 B	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		1.6E−01 B	8.9E−02 B					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				4E−06 W	8E−06 W					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						1E−08 B	5E−08 B					NA	NA

B – below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1×10^{-6})
W – within EPA target cancer risk range (ELCR>1×10^{-6} and <1×10^{-4})
B – exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1×10^{-4})

Noncancer Effects

Open Land Use. Tables 5.42 through 5.44 indicate that the total HI scores for combined exposure across pathways for open land use at the SLB fall below the target established by EPA (i.e., HI is less than 1).

Cancer Effects

Open Land Use. The cancer risks for chemical carcinogens are all on the order of 10^{-6}. The radionuclide cancer risks are nearly two orders of magnitude lower (10^{-8}) than the chemical cancer risks. The combined cancer risks are within the EPA target cancer risk range. The soil ingestion route dominates, and all other estimates are below the EPA target range.

Arsenic is the only substance contributing significantly to the cancer risk estimate, accounting for nearly all of the cancer risk.

5.4.4.3 EPA risk characterization method for lead exposures

No toxicity values (e.g., RfDs and CSFs) are currently available from EPA for lead exposures, and EPA has withdrawn the former RfD for lead. Considerable uncertainty exists regarding the selection of a single point estimate for acceptable or unacceptable health effects due to lead. As noted in more detail in Sect. 5.3, recent toxicologic developments indicate that health effects attributable to lead may occur at much lower exposures than previously thought. Furthermore, these health effects may or may not be associated with a distinct threshold below which there are *no* adverse effects. In particular, mutagenic reproductive effects, neurological effects, and learning disabilities in children may occur at relatively low exposure levels. The evidence has prompted a reduction in the latest Centers for Disease Control (CDC) guideline for blood lead levels in children, from 25 to 10 μg/dL lead.

The risk characterization for lead was based on an alternative approach developed by EPA, in which lead uptake into children's blood is estimated. Blood lead levels are considered a more appropriate indicator of low-level lead exposures than are the classic symptoms associated with higher-level lead exposure. EPA has developed a computer program, LEAD 0.60 (EPA 1991e), to study blood lead uptake from various environmental sources. LEAD 0.60 is an uptake biokinetic model that allows the computer operator to estimate blood lead uptake in children, the most sensitive receptors of lead exposures. The lead model is not currently applicable to adults, so analysis using LEAD 0.60 was limited to the land uses and areas of EFPC that included children as potential receptors.

All of the chemicals evaluated at EFPC, including lead, relate to *excess* chemical exposures, which are those site-related exposures above and beyond those encountered off site. As was the case for the other chemicals, background levels of lead at the study areas are included in the exposure point concentrations.

LEAD 0.60 is designed to accept either adjusted or default inputs. For the purpose of this discussion, the adjustments fall into two distinct types: those that affect the underlying mechanism of the model, and exposure parameters describing conditions at EFPC, such as ingestion rate and frequency. In the evaluation of lead at EFPC, only the latter exposure parameters were adjusted in the LEAD 0.60 model. No adjustment was made to the default absorption methodology used in the model; the model was run using the unmodified nonlinear active-passive absorption methodology. Furthermore, the model uses the recommended default (unadjusted) geometric standard deviation (GSD = 1.42) for the distribution of estimated blood lead levels.

In order to be consistent with the exposures considered in the BRA, an understanding of the effect of the built-in default exposure parameters (i.e., 0.2 $\mu g/m^3$ for inhalation, various dietary lead intakes from food sources) is essential. Default exposure parameters are provided in the model as reasonable defaults to assist the operator in a wide variety of situations where site-specific data are lacking. Although such defaults could be important in understanding the total hypothetical body burden of lead, they do not always enhance the stated purpose of the BRA, since they do not pertain to site-related sources of lead. Consideration and adjustment of the appropriate default exposure parameters (although not the underlying mechanism of the model) is therefore essential to support the risk assessment process.

Evaluating Lead Exposures at EFPC. Lead was detected in soil samples at RME exposure point concentrations ranging from 32.3 mg/kg at Segment 6 to 136 mg/kg at Segment 4. These soil lead exposure point concentrations are based on segment-wide aggregations of the available data. Background soil samples contained lead at 14.1 mg/kg. Lead was detected in all soil samples that were analyzed for lead. Table P.1 in Appendix P indicates that most (essentially all) of the segment soil samples exceeded the background tolerance interval for lead (as defined by samples from Hinds Creek). Lead thus appears to exceed concentrations expected for background soils at each segment. Lead was detected in groundwater at an RME concentration of 38.3 (total) and 1.7 ug/L (dissolved). Note that a single groundwater data set represents all segments, and that no background data are available for groundwater.

Table 5.45 presents the exposure point concentrations for lead at each segment and notes key exposure assumptions used in the lead model. Exposure assumptions were set to match as much

**Table 5.45. LEAD 0.6 Uptake/Biokinetic Model:
Current and Future Exposure Point Concentrations and Assumptions**

Segment	Current Land Use (excluding foodchain)	Future Land Use (excluding foodchain and using dissolved lead concentrations in groundwater)	Future Land Use (excluding foodchain and using total lead concentrations in groundwater)
1	Soil - 47.7 mg/kg Air - 1.03 x 10^{-5} $\mu g/m^3$	Soil - 47.7 mg/kg Air - 1.03 x 10^{-5} $\mu g/m^3$ Water - 1.69 $\mu g/L$	Soil - 47.7 mg/kg Air - 1.03 x 10^{-5} $\mu g/m^3$ Water - 38.3 $\mu g/L$
2	Soil - 55.9 mg/kg Air - 1.21 x 10^{-5} $\mu g/m^3$	Soil - 55.9 mg/kg Air - 1.21 x 10^{-5} $\mu g/m^3$ Water - 1.69 $\mu g/L$	Soil - 55.9 mg/kg Air - 1.21 x 10^{-5} $\mu g/m^3$ Water - 38.3 $\mu g/L$
3	Soil - 89.5 mg/kg Air - 1.93 x 10^{-5} $\mu g/m^3$	Soil - 89.5 mg/kg Air - 1.93 x 10^{-5} $\mu g/m^3$ Water - 1.69 $\mu g/L$	Soil - 89.5 mg/kg Air - 1.93 x 10^{-5} $\mu g/m^3$ Water - 38.3 $\mu g/L$
4	Soil - 136 mg/kg Air - 2.94 x 10^{-5} $\mu g/m^3$	Soil - 136 mg/kg Air - 2.94 x 10^{-5} $\mu g/m^3$ Water - 1.69 $\mu g/L$	Soil - 136 mg/kg Air - 2.94 x 10^{-5} $\mu g/m^3$ Water - 38.3 $\mu g/L$
5	Soil - 72.2 mg/kg Air - 1.56 x 10^{-5} $\mu g/m^3$	Soil - 72.2 mg/kg Air - 1.56 x 10^{-5} $\mu g/m^3$ Water - 1.69 $\mu g/L$	Soil - 72.2 mg/kg Air - 1.56 x 10^{-5} $\mu g/m^3$ Water - 38.3 $\mu g/L$
6	Soil - 32.3 mg/kg Air - 6.98 x 10^{-6} $\mu g/m^3$	Soil - 32.3 mg/kg Air - 6.98 x 10^{-6} $\mu g/m^3$ Water - 1.69 $\mu g/L$	Soil - 32.3 mg/kg Air - 6.98 x 10^{-6} $\mu g/m^3$ Water - 38.3 $\mu g/L$
7	Soil - 43.7 mg/kg Air - 9.44 x 10^{-6} $\mu g/m^3$	Soil - 43.7 mg/kg Air - 9.44 x 10^{-6} $\mu g/m^3$ Water - 1.69 $\mu g/L$	Soil - 43.7 mg/kg Air - 9.44 x 10^{-6} $\mu g/m^3$ Water - 38.3 $\mu g/L$
8	Soil - 77.8 mg/kg Air - 1.68 x 10^{-5} $\mu g/m^3$	Soil - 77.8 mg/kg Air - 1.68 x 10^{-5} $\mu g/m^3$ Water - 1.69 $\mu g/L$	Soil - 77.8 mg/kg Air - 1.68 x 10^{-5} $\mu g/m^3$ Water - 38.3 $\mu g/L$
9	Soil - 40.3 mg/kg Air - 8.70 x 10^{-6} $\mu g/m^3$	Soil - 40.3 mg/kg Air - 8.70 x 10^{-6} $\mu g/m^3$ Water - 1.69 $\mu g/L$	Soil - 40.3 mg/kg Air - 8.70 x 10^{-6} $\mu g/m^3$ Water - 38.3 $\mu g/L$
Background	Soil - 14.1 mg/kg Air - 3.05 x 10^{-6} $\mu g/m^3$	Soil - 14.1 mg/kg Air - 3.05 x 10^{-6} $\mu g/m^3$ Water - 1.69 $\mu g/L$	Soil - 14.1 mg/kg Air - 3.05 x 10^{-6} $\mu g/m^3$ Water - 38.3 $\mu g/L$

The particulate emission factor (PEF) ,4.63 x 10^9 kg/m^3 (RAGS, Part B), was used to estimate airborne lead concentrations. Lead concentrations in drinking water for current land use assumptions were treated as zero, and groundwater monitoring results were substituted for the default concentrations under future land use assumptions. Default dietary lead intakes were used for estimating blood lead levels for future scenarios. Soil monitoring results were substituted for default soil lead levels. The soil intake rate was set at 200 mg/d, and the soil/dust weighting factor at 100 % (all soil).

as possible the residential exposure assumptions used in the risk assessment for other chemicals. Food chain exposures was not included because of the large uncertainties identified for the food chain pathway. The lead model was run several ways to evaluate the sensitivity of combining site data for soil and groundwater: using both soil and groundwater exposure point concentrations, and excluding groundwater exposure point concentrations. In addition, exposures to both unfiltered and filtered groundwater also are included in the evaluation. The soil and groundwater exposures are not simply additive in the lead model; combined soil and groundwater exposures result in higher blood lead levels than simply adding separate blood lead estimates for soil and groundwater.

When interpreting the LEAD 0.60 model results, note that the CDC guideline discussed above is the current EPA benchmark for evaluating lead exposures. Exposure limits are further defined by a maximum of 5% of the exposed population exceeding the CDC blood lead cutoff. This is based on EPA guidance for an appropriate point of reference (EPA 1991e):

> "We recommend a model projection benchmark of either 95% of the sensitive population having blood lead levels below 10 μg/dL or a 95% probability of an individual having a blood lead level below 10 μg/dL."

Lead exposures are thus defined as within the EPA target if no more than 5% of the modeled population exceeds the CDC blood lead cutoff.

Results of Risk Characterization for Lead Exposures. The LEAD 0.60 results as generated by the model are presented in Appendix M. These results are summarized below in Tables 5.46 through 5.48. As noted above, the LEAD 0.60 model is not designed for analysis of adults. Under current land use assumptions, only Segments 3, 6, 7, and 8 include exposures to children. Table 5.46 indicates that none of the blood lead levels associated with current exposures at any of these segments approach the CDC blood lead cutoff.

When considering future exposures, blood lead levels are generally within the noted EPA target. Table 5.47 indicates that under future land use, blood lead levels for a child residing at Segments 3 and 4 exceed the EPA target. The target was exceeded only when simultaneously combining both soil and groundwater exposures. The exceedance is marginal and must be qualified with the understanding that the results shown in Table 5.47 combine soil, unfiltered groundwater, and default exposure assumptions for both the air pathway and nonsite-related dietary sources of lead.

Table 5.47 also includes a comparison of the combined results, with the substitution of unfiltered versus filtered groundwater exposures. Only unfiltered groundwater exposures are

Table 5.46. LEAD 0.60 Uptake/Biokinetic Model Results: Current Land Use[a] Contribution from Soil and Air

	Segment 1	Segment 2	Segment 3	Segment 4	Segment 5	Segment 6	Segment 7	Segment 8	Segment 9
Percentile Exceeding 10 μg Pb/dL Blood Lead Level	--	--	0.00	--	--	0.00	0.00	0.00	--
Geometric Mean of the Projected Blood Lead Level (μg Pb/dL blood)	--	--	2.60	--	--	1.60	1.80	2.40	--
Sensitive Age Group (months)	--	--	72 to 84	--	--	72 to 84	72 to 84	72 to 84	--

-- Children 0-7 years were not evaluated in these segments (the land use in these segments is open and focuses on an older child).

[a] Results were estimated using default LEAD 0.60 intake assumptions with the following exceptions:

- the particulate emission factor, in conjunction with measured lead concentrations in soil, was used to calculate airborne lead concentrations (10^{-7} to 10^{-4} μg/m^3) in place of the default setting (0.200 μg/m^3),
- lead concentrations in drinking water were treated as zero,
- soil monitoring results were substituted for soil Pb levels, the amount of soil ingested daily was set at 0.2 g/day, and the soil/dust weighting factor was set at 100 % (all soil).

Table 5.47. LEAD 0.60 Uptake/Biokinetic Model Results: Future Land Use[a]
Contribution from Soil, Groundwater, and Air
(Total and Dissolved Pb Concentrations)

Total Pb

	Segment 1	Segment 2	Segment 3	Segment 4	Segment 5	Segment 6	Segment 7	Segment 8	Segment 9
Percentile Exceeding 10 μg Pb/dL Blood Lead Level	2.70	3.12	5.92	11.84	4.46	1.75	2.34	4.79	2.18
Geometric Mean of the Projected Blood Lead Level (μg Pb/dL blood)	5.12	5.26	5.86	6.68	5.55	4.85	5.05	5.65	4.99
Sensitive Age Group (months)	72 to 84	72 to 84	72 to 84	72 to 84	72 to 84	72 to 84	72 to 84	72 to 84	72 to 84

Dissolved Pb

	Segment 1	Segment 2	Segment 3	Segment 4	Segment 5	Segment 6	Segment 7	Segment 8	Segment 9
Percentile Exceeding 10 μg Pb/dL Blood Lead Level	0.00	0.00	0.01	0.19	0.00	0.00	0.00	0.00	0.00
Geometric Mean of the Projected Blood Lead Level (μg Pb/dL blood)	2.01	2.16	2.75	3.57	2.44	1.74	1.94	2.54	1.88
Sensitive Age Group (months)	72 to 84	72 to 84	72 to 84	72 to 84	72 to 84	72 to 84	72 to 84	72 to 84	72 to 84

[a] Results were estimated using default LEAD 0.60 intake assumptions with the following exceptions:

- the particulate emission factor, in conjunction with measured lead concentrations in soil, was used to calculate airborne lead concentrations (10^{-7} to 10^{-4} μg/m^3) in place of the default setting (0.200 μg/m^3),
- groundwater monitoring results (total and dissolved) were substituted for the lead concentration in drinking water,
- soil monitoring results were substituted for soil Pb levels, the amount of soil ingested daily was set at 0.2 g/day, and the soil/dust weighting factor was set at 100 % (all soil).

Table 5.48. LEAD 0.60 Uptake/Biokinetic Model Results: Future Land Use[a]
Contribution from Soil and Air

	Segment 1	Segment 2	Segment 3	Segment 4	Segment 5	Segment 6	Segment 7	Segment 8	Segment 9
Percentile Exceeding 10 μg Pb/dL Blood Lead Level	0.00	0.00	0.00	0.10	0.00	0.00	0.00	0.00	0.00
Geometric Mean of the Projected Blood Lead Level (μg Pb/dL blood)	1.87	2.01	2.60	3.42	2.30	1.60	1.80	2.40	1.74
Sensitive Age Group (months)	72 to 84	72 to 84	72 to 84	72 to 84	72 to 84	72 to 84	72 to 84	72 to 84	72 to 84

[a] Results were estimated using default LEAD 0.60 intake assumptions with the following exceptions:

- the particulate emission factor, in conjunction with measured lead concentrations in soil, was used to calculate airborne lead concentrations (10^{-7} to 10^{-4} μg/m^3) in place of the default setting (0.200 μg/m^3),
- lead concentrations in drinking water were treated as zero,
- soil monitoring results were substituted for soil Pb levels, the amount of soil ingested daily was set at 0.2 g/day, and the soil/dust weighting factor was set at 100 % (all soil).

associated with blood lead levels exceeding the EPA target. Table 5.48 includes soil and default air pathway exposures, and shows that when groundwater exposures are excluded, the remaining exposures yield blood lead level estimates well below the EPA target.

Since use of the LEAD 0.60 model is not applicable for adults, an additional point of comparison may be useful. The soil lead exposure point concentrations may be compared to recently published soil lead guidelines (EPA 1991g). The guidelines establish a range of 500 to 1000 mg/kg total lead in soil as interim cleanup levels; the soil lead exposure point concentrations at each segment fall substantially below these guidelines. Like LEAD 0.60, the soil lead guidelines were developed for residential settings, with a particular focus on children. The use of these guidelines is less suited to occupational or workplace settings, since workers spend less time on site than residents, and children generally are not present. Consequently, future land use becomes an important issue to consider when comparing lead levels at EFPC to these conservative guidelines.

5.4.4.4 Comparisons with background

In this section, comparison is presented for background concentration of chemicals at the Hinds Creek reference site floodplain contaminant levels in EFPC floodplain soil. A tolerance interval approach is used and sample by sample comparison is conducted. As an additional tool for interpreting the results of the Tier II analysis, risk estimates are presented using background contaminant concentrations (from Hinds Creek) and the agricultural/homesteader exposure assumptions. Tables presenting the results of the tolerance interval and risk estimate comparison to background are found in Appendix P.

The objectives of the background assessment are to understand the significance of background levels, further explore conservatism built into the exposure assumptions, evaluate risk from soil levels at the background location, and compare these risks to risks from soil levels in the floodplain.

Tolerance Interval Comparison for Segment 1

Noncancer Effects. The four substances of greatest concern for noncancer effects in Segment 1 soils (i.e., arsenic, cadmium, manganese, and mercury) were evaluated against values for these substances detected during background testing at Hinds Creek. Where background data were sufficiently robust, results from testing in Segment 1 were compared against background values using the tolerance interval method at 95% confidence. Values for arsenic were statistically significant in 56 of 101 sample comparisons, indicating that the data for arsenic in soil at Segment 1 exceeded that expected in the reference area, and may provide evidence of site

contamination. Values for manganese in Segment 1 were not statistically significant in any of the 17 comparisons.

Tolerance interval tests were not possible for mercury or cadmium because of the limited number of samples collected with concentrations above detection limits at the Hinds Creek background segment. Cadmium was not detected in any of 12 samples, and mercury was detected in only 1 of 12 samples taken during background testing at Hinds Creek. Segment 1 testing identified cadmium concentrations above detection limits in 12 of 17 samples and mercury concentrations above detection limits in 51 of 94 samples. These results are above that expected as determined for the reference area, and may thus indicate site-related contamination by these substances.

Cancer Effects. The two substances of greatest concern for cancer effects in Segment 1 (i.e., arsenic and beryllium) were evaluated against values for these substances found during background testing at Hinds Creek. Results from testing at Segment 1 were compared against background values using the tolerance interval method at 95% confidence. Values for arsenic were statistically significant in 56 of 101 comparisons, in that they appear to exceed the reference area concentrations. The arsenic data from Segment 1 may thus indicate site-related contamination. Values for beryllium were statistically significant in only 3 of 17 comparisons. These data indicate that beryllium contamination may not be site related.

Tolerance Interval Comparison for Segment 2

Noncancer Effects. Arsenic, cadmium, manganese, and mercury from Segment 2 were evaluated against values for these substances found during background testing at Hinds Creek. Where background data were sufficiently robust, results from testing in Segment 2 were compared against background values using the tolerance interval method at 95% confidence. Values for arsenic were statistically significant in 83 of 98 comparisons; these data are above that expected in the reference area, and may be site related. Values for manganese were not statistically significant in any of the 17 comparisons.

Tolerance interval tests were not possible for mercury or cadmium because of the limited number of samples collected with concentrations above detection limits at the Hinds Creek background segment. Cadmium was not detected in any of 12 samples, and mercury was detected in only 1 of 12 samples collected during background testing at Hinds Creek. Segment 2 testing identified cadmium concentrations above detection limits in all 6 samples tested, and mercury concentrations above detection limits in 27 of 83 samples. Since these results

significantly exceed the reference area results, there may be evidence of site-related contamination by these substances.

Cancer Effects. The two substances of greatest concern for cancer effects in Segment 2 (i.e., arsenic and beryllium) were evaluated against values for these substances found during background testing at Hinds Creek. Results from testing in Segment 2 were compared against background values using the tolerance interval method at 95% confidence. Values for arsenic were statistically significant in 83 of 98 comparisons, which means these data provide evidence of site-related contamination. However, values for beryllium were statistically significant in none of the six comparisons, indicating no site-related beryllium contamination.

Tolerance Interval Comparison for Segment 3

Noncancer Effects. The four substances of greatest concern for noncancer effects in Segment 3 (i.e., arsenic, cadmium, manganese, and mercury) were evaluated against values for these substances found during background testing at Hinds Creek. Where background data were sufficiently robust, results from testing in Segment 3 were compared against background values using the tolerance interval method at 95% confidence. Values for arsenic were statistically significant in 64 of 70 comparisons, indicating that these data exceed expected levels and may thus be site related. Values for manganese were not statistically significant in any of the 14 comparisons.

Tolerance interval tests were not possible for mercury or cadmium because of the limited number of samples collected with concentrations above detection limits at the Hinds Creek background segment. Cadmium was not detected in any of 12 samples, and mercury was detected in only 1 of 12 samples collected during background testing at Hinds Creek. Segment 3 sample results identified cadmium concentrations above detection limits in 8 of 14 samples and mercury concentrations above detection limits in 36 of 72 samples. These results may thus indicate site-related contamination by these substances.

Cancer Effects. The two substances of greatest concern for cancer effects in Segment 3 (i.e., arsenic and beryllium) were evaluated against values for these substances detected during background testing at Hinds Creek. Results from testing in Segment 3 were compared against background values using the tolerance interval method at 95% confidence. Values for arsenic were statistically significant in 64 of 70 comparisons, and suggest these data may be site related. Values for beryllium were statistically significant in none of the 14 comparisons; these data do not support an indication of site-related contamination.

Tolerance Interval Comparison for Segment 4

Noncancer Effects. The four substances of greatest concern for noncancer effects in Segment 4 (i.e., arsenic, cadmium, manganese, and mercury) were evaluated against values for these substances found during background testing at Hinds Creek. Where background data were sufficiently robust, results from testing in Segment 4 were compared against background values using the tolerance interval method at 95% confidence. Values for arsenic were statistically significant in 95 of 100 comparisons; these data are clearly site related. Values for manganese were not statistically significant in any of the nine comparisons.

Tolerance interval tests were not possible for mercury or cadmium because of the limited number of samples collected with concentrations above detection limits at the Hinds Creek background segment. Cadmium was not detected in any of 12 samples, and mercury was detected in only 1 of 12 samples collected during background testing at Hinds Creek. Segment 4 testing identified cadmium concentrations above detection limits in 8 of 9 samples and mercury concentrations above detection limits in 69 in 109 samples. These results may indicate site-related contamination by these substances.

Cancer Effects. Cancer risk estimates were not of concern in Segment 4. However, a background comparison was conducted for the two substances of potential concern for cancer effects (i.e., arsenic and beryllium). Concentrations of the chemicals were evaluated against levels found during background testing at Hinds Creek. Results from testing in Segment 4 were compared against background values using the tolerance interval method at 95% confidence. Values for arsenic were statistically significant in 95 of 100 comparisons, suggesting that these data may be site related. Values for beryllium were statistically significant in only three of nine comparisons. These data do not support an indication that beryllium is a site-related contaminant.

Tolerance Interval Comparison for Segment 5

Noncancer Effects. The four substances of greatest concern for noncancer effects in Segment 5 (i.e., arsenic, cadmium, manganese, and mercury) were evaluated against values for these substances detected during background testing at Hinds Creek. Where background data were sufficiently robust, results from testing in Segment 5 were compared against background values using the tolerance interval method at 95% confidence. Values for arsenic were statistically significant in 67 of 72 comparisons; these data may indicate site-related contamination. Values for manganese were not statistically significant in any of the six comparisons.

Tolerance interval tests were not possible for mercury or cadmium because of the limited number of samples collected with concentrations above detection limits at the Hinds Creek background segment. Cadmium was not detected in any of 12 samples, and mercury was detected in only 1 of 12 samples collected during background testing at Hinds Creek. Segment 5 testing identified cadmium concentrations above detection limits in 5 of 6 samples and mercury concentrations above detection limits in 35 of 72 samples. These results may indicate site-related contamination by these substances.

Cancer Effects. The two substances of greatest concern for cancer effects in Segment 5 (i.e., arsenic and beryllium) were evaluated against values for these substances detected during background testing at Hinds Creek. Results from testing in Segment 5 were compared against background values using the tolerance interval method at 95% confidence. Values for arsenic were statistically significant in 67 of 72 comparisons, providing evidence that these data may be site related. However, values for beryllium were statistically significant in none of the six comparisons, in fact none of the values was above detection limits. These results do not support the indication that there is site-related contamination by beryllium.

Tolerance Interval Comparison for Segment 6

Noncancer Effects. The four substances of greatest concern for noncancer effects in Segment 6 (i.e., arsenic, cadmium, manganese, and mercury) were evaluated against values for these substances detected during background testing at Hinds Creek. Where background data were sufficiently robust, results from testing in Segment 6 were compared against background values using the tolerance interval method at 95% confidence. Values for arsenic were statistically significant in 44 of 62 comparisons. These data suggest that there may be evidence of site-related contamination for arsenic. Values for manganese were not statistically significant in any of the seven comparisons.

Tolerance interval tests were not possible for mercury or cadmium because of the limited number of samples collected with concentrations above detection limits at the Hinds Creek background segment. Cadmium was not detected in any of 12 samples, and mercury was detected in only 1 of 12 samples collected during background testing at Hinds Creek. Segment 6 testing identified cadmium concentrations above detection limits in 2 of 7 samples and mercury concentrations above detection limits in 28 of 63 samples. These results may indicate site-related contamination by these compounds.

Cancer Effects. The substance of greatest concern for cancer effects in Segment 6 (i.e., arsenic) was evaluated against values for this substance detected during background testing

at Hinds Creek. Results from testing in Segment 6 were compared against background values using the tolerance interval method at 95% confidence. Values for arsenic were statistically significant in 44 of 62 comparisons, and may provide evidence of site-related contamination.

Tolerance Interval Comparison for Segment 7

Noncancer Effects. The four substances of greatest concern for noncancer effects in Segment 7 (i.e., arsenic, cadmium, manganese, and mercury) were evaluated against values for these substances detected during background testing at Hinds Creek. Where background data were sufficiently robust, results from testing in Segment 7 were compared against background values using the tolerance interval method at 95% confidence. Values for arsenic were statistically significant in 257 of 291 comparisons; these data may indicate site-related contamination. Values for manganese were statistically significant in only one of eight comparisons.

Tolerance interval tests were not possible for mercury or cadmium because of the limited number of samples collected with concentrations above detection limits at the Hinds Creek background segment. Cadmium was not detected in any of 12 samples, and mercury was detected in only 1 of 12 samples collected during background testing at Hinds Creek. Segment 7 testing identified cadmium concentrations above detection limits in 4 of 8 samples and mercury concentrations above detection limits in 84 of 326 samples. These results are evidence of site-related contamination by these chemicals.

Cancer Effects. The substance of greatest concern for cancer effects in Segment 7 (i.e., arsenic) was evaluated against values for this substance detected during background testing at Hinds Creek. Results from testing in Segment 7 were compared against background values using the tolerance interval method at 95% confidence. Values for arsenic were statistically significant in 257 of 291 comparisons, and may thus be site related.

Tolerance Interval Comparison for Segment 8

Noncancer Effects. The four substances of greatest concern for noncancer effects in areas of residential and commercial land uses in Segment 8 (i.e., arsenic, cadmium, manganese, and mercury) were evaluated against values for these substances detected during background testing at Hinds Creek. Where background data were sufficiently robust, results from testing in Segment 8 were compared against background values using the tolerance interval method at 95% confidence. Values for arsenic were statistically significant in 69 of 85 comparisons; these data exceed expected background levels. While the mean value for Segment 8 manganese

concentrations was roughly 70% above the mean detected at Hinds Creek, values for manganese were not statistically significant in any of the eight comparisons.

Tolerance interval tests were not possible for mercury or cadmium because of the limited number of samples collected with concentrations above detection limits at the Hinds Creek background segment. Cadmium was not detected in any of 12 samples, and mercury was detected in only 1 of 12 samples taken during background testing at Hinds Creek. Segment 8 testing identified cadmium concentrations above detection limits in 6 of 8 samples and mercury concentrations above detection limits in 37 of 85 samples. Even without clear indications from the tolerance interval test, these substances exceed background levels.

Cancer Effects. The substance of greatest concern for cancer effects in Segment 8 (i.e., arsenic) was evaluated against values for this substance detected during background testing at Hinds Creek. Results from testing in Segment 8 were compared against background values using the tolerance interval method at 95% confidence. Values for arsenic were statistically significant in 69 of 85 comparisons; these data clearly exceed background.

Tolerance Interval Comparison for Segment 9

Noncancer Effects. The four substances of greatest concern for noncancer effects in areas of residential land use in Segment 9 (i.e., arsenic, cadmium, manganese, and mercury) were evaluated against values for these substances detected during background testing at Hinds Creek. Where background data were sufficiently robust, results from testing in Segment 9 were compared against background values using the tolerance interval method at 95% confidence. Values for arsenic were statistically significant in 227 of 320 comparisons; these data exceed levels expected in the background segment and may be site related. While the mean value for Segment 9 manganese concentrations was roughly 85% above the mean found at Hinds Creek, values for manganese were not statistically significant in any of the 18 comparisons.

Tolerance interval tests were not possible for mercury or cadmium because of the limited number of samples collected with concentrations above detection limits at the Hinds Creek background segment. Cadmium was not detected in any of 12 samples, and mercury was detected in only 1 of 12 samples collected during background testing at Hinds Creek. Segment 9 testing identified cadmium concentrations above detection limits in 14 of 18 samples and mercury concentrations above detection limits in 153 of 697 samples. These results indicate contamination above background levels by these compounds.

Cancer Effects. The substance of greatest concern for cancer effects in Segment 9 (i.e., arsenic) was evaluated against values for this substance detected during background testing at Hinds Creek. Results from testing in Segment 9 were compared against background values using the tolerance interval method at 95% confidence. Values for arsenic were statistically significant in 227 of 320 comparisons; these data clearly exceed background.

Risk Estimate Comparison of EFPC and Hinds Creek

Risk characterization tables were prepared for the background analysis at Hinds Creek and are presented in Appendix P. These results are summarized in Table 5.49.

Noncancer Effects. The results of the background analysis show the HIs and cancer risks for Hinds Creek are well above EPA's noncancer target benchmark of 1.0 and the upper limit of the excess lifetime cancer risk range of 10^{-6} to 10^{-4}. It is particularly important to note that this occurs despite the fact that the background samples were collected from an area not affected by releases from the Y-12 Plant. The individual pathways principally associated with the high HI scores are the food chain pathways, as was the case with the Tier I and Tier II results. Despite the fact that no mercury contamination is present in the background soils, the produce, beef, and dairy ingestion pathways still each show HI scores that are above the EPA noncancer limit of 1.

For the produce and beef ingestion pathways, arsenic and manganese are related to more than 90% of the risk. For the dairy ingestion pathway, arsenic, barium, copper, manganese, and zinc are associated with more than 90% of the risk. Risks from inadvertent soil ingestion and dermal contact are within the target limits established by EPA for both adults and children, with HI scores below 1.

Cancer Effects. The total cancer risks associated for all pathways combined are well above EPA's target risk-range despite the fact that these samples were collected from the Hinds Creek background location. As was the case for noncancer effects, the food chain pathways are responsible for the majority of the risk. Risks from ingestion of contaminated beef are the highest, although the risks from the produce and dairy ingestion pathways also are on the order of 10^{-4}. Cancer risks related to fish ingestion are on the order of 10^{-5}, which is within the EPA target range. The soil ingestion pathway cancer risks also fall within the EPA target range. In all cases except fish ingestion, arsenic is the primary risk driver. For the fish ingestion pathway, PCBs are responsible for all of the risk.

Table 5.49. Risk characterization summary for Hinds Creek: RME estimates for the future land uses

		AGRICULTURAL LAND USE						COMMERCIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	1.03E−01 B	2.88E−02 B	5.29E−06 W	4.92E−06 W	1.24E−08 B	3.10E−08 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	4.10E−03 B	3.13E−03 B	2.10E−07 B	5.35E−07 B	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	5.62E−03 B	2.09E−03 B	2.60E−07 B	3.22E−07 B	7.01E−07 B	7.01E−07 B	NA	NA	NA	NA	NA	NA
	Dermal Contact	1.86E−03 B	1.43E−03 B	7.10E−08 B	1.81E−07 B	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	2.63E−04 B	2.01E−04 B	1.01E−08 B	2.59E−08 B	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	9.69E+00 *B*	4.67E+00 *B*	1.83E−04 *B*	2.94E−04 *B*	1.80E−07 B	7.78E−07 B	NA	NA	NA	NA	NA	NA
Beef	Ingestion	2.77E+01 *B*	1.39E+01 *B*	1.71E−03 *B*	2.87E−03 *B*	2.76E−07 B	1.25E−06 W	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	1.22E+01 *B*	2.94E+00 *B*	2.15E−04 *B*	1.73E−04 *B*	8.64E−07 B	1.88E−06 W	NA	NA	NA	NA	NA	NA
Fish	Ingestion	2.75E−02 B	1.32E−02 B	1.04E−05 W	1.66E−05 W	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	9.02E−02	NA	8.80E−07	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		5.0E+01 *B*	2.2E+01 *B*					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				2E−03 *B*	3E−03 *B*					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						2E−06 W	5E−06 W					NA	NA

		OPEN LAND USE						RESIDENTIAL LAND USE					
		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)		NONCANCER		CANCER		RADIONUCLIDES (CANCER ONLY)	
MEDIA	ROUTE	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT	CHILD	ADULT
Soil	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Surface Water	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Sediment	Dermal Contact	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Groundwater	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	Inhalation (VOCs)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Produce	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Beef	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Dairy	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Fish	Ingestion	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Air	Particulates: Harvesting	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Chemical Hazards: Hazard Index (HI): Combined Exposure		NA	NA					NA	NA				
Excess Lifetime Cancer Risk: Combined Exposure				NA	NA					NA	NA		
Radiological Hazards: Excess Lifetime Cancer Risk: Combined Exposure						NA	NA					NA	NA

B – below or equal to target noncancer hazard index (HI<=1), or cancer risk (ELCR<=1 x 10^{-6})
W – within EPA target cancer risk range (ELCR>1 x 10^{-6} and <1 x 10^{-4})
B – exceeds target noncancer hazard index (HI>1), or cancer risk (ELCR>1 x 10^{-4})

Radionuclide cancer risks represent less than 1% of the total cancer risk. Ingestion of beef and dairy products remain the pathways representing the majority of the risk. Within these pathways, cesium 137+D and cobalt 60 are associated with the highest cancer risks.

In summary, conducting risk assessment on the background samples collected at Hinds Creek has served to emphasize the conservative nature of the exposure assumptions for the food chain pathways. In particular, the cancer risk estimates and HI scores for the reference area at Hinds Creek exceed EPA target risk ranges for an area that is not affected by releases from the Y-12 Plant.

5.4.4.5 Summary of Tier II analysis: creek-wide human health risk assessment

Key Features of Tier II Risk Assessment

The Tier II BRA is an evaluation of the potential risks to human health within the floodplain area of all nine segments along EFPC. This assessment uses monitoring data from both Phases Ia and Ib. The risk assessment considers both current and future land uses, and examines the potential for adverse health effects in adults and children. Environmental media and exposure pathways include soil, groundwater, surface water, sediment, and food chain. The Tier II risk assessment focuses primarily on upper-bound RME risks, although risks based on more central estimates (MLE) are also available (Appendix M).

Results of Tier II Risk Assessment

Sections 5.4.3.2 and 5.4.3.3 present a detailed discussion of the results of the human health risk assessment for each land use scenario and exposure pathway. The results are based on the RME exposure assumptions outlined in the exposure assessment section (Sect. 5.2). This section of the BRA provides a summary and discussion of the many analyses used in the Tier II human health risk assessment.

Reviewing Tables 5.20 through 5.41, a number of principal findings may be reported. The residential and agricultural scenarios are associated with the greatest human health risks, which exceed of EPA targets for both current and future land use assumptions. The open land use scenarios never resulted in human health risks exceeding EPA targets for either current or future land uses. Health risks associated with the commercial land use scenario exceeded EPA limits, but this was entirely due to hypothetical groundwater ingestion. The greatest health risks were associated with exposure to chemical contaminants, and all of the risks for radionuclides fell within the targets established by EPA.

The results of the BRA show that the exposure pathways of primary concern for the residential and agricultural scenarios are: inadvertent ingestion exposure to soils; food chain pathways that involve the movement of contaminants from soil to produce, beef, and dairy products; and hypothetical use of groundwater as a source of drinking water. Health effects targets established by EPA were exceeded in each of these exposure pathways. Ingestion of fish and recreational use of EFPC (i.e., use of the creek for swimming and wading) fell within or below targets established by EPA.

Soil Exposures. The principal concern for the soil ingestion pathway is noncancer effects associated with exposure to mercury. Chemicals contributing a lesser degree to the HI score include cadmium, arsenic, and manganese. Cancer risk estimates associated with inadvertent ingestion of soils all fell within the target range established by EPA. Noncancer risks related to soil ingestion exceeded the target range established by EPA in the following segments:

- Segment 1: future resident children, noncancer only,
- Segment 3: current and future resident children, noncancer only,
- Segment 4: future resident children and adults, noncancer only,
- Segment 5: future resident children, noncancer only, and
- Segment 8: current and future resident children, noncancer only.

Food Chain Exposures. Risks estimates associated with the food chain pathways greatly exceeded the target range established by EPA for waste site remediation. This is true for all segments and land use areas for which residential and agricultural scenarios are included in the risk analysis. The residential scenario evaluated ingestion of contaminated produce obtained from home gardens grown in floodplain soils and ingestion of fish caught from EFPC (recreational fishing). The agricultural scenario examined ingestion of beef and dairy products in addition to the produce pathway and ingestion of fish. The pathways of concern (i.e., those resulting in elevated risks) are ingestion of produce, beef, and dairy products. Risks associated with exposure to foods produced in the floodplain area at EFPC exceeded the target range established by EPA in the following segments:

- Segment 1: future resident children and adults, noncancer and cancer,
- Segment 2: future resident children and adults, noncancer and cancer,
- Segment 3: current and future resident children and adults, noncancer and cancer,
- Segment 4: future resident children and adults, noncancer and cancer,
- Segment 5: future resident children and adults, noncancer and cancer,
- Segment 6: current and future resident children and adults, noncancer and cancer,
- Segment 7: current and future agricultural children and adults, noncancer and cancer,
- Segment 8: current and future resident children and adults, noncancer and cancer, and
- Segment 9: future agricultural and resident children and adults, noncancer and cancer.

The produce pathway accounted for the greatest percentage of risk to human health. For noncancer effects, four contaminants (i.e., mercury, manganese, cadmium, and arsenic) are principally responsible for these risks. For cancer effects, arsenic was the only substance exceeding EPA targets.

Groundwater Exposures. Risk estimates for hypothetical use of groundwater as drinking water exceeded the target range established by EPA. The groundwater exposures are presented as part of the future land use analysis for the agricultural, residential, and commercial scenarios (Sect. 5.4.3.3 and Appendix M). The risk estimates for the groundwater pathway, regardless of creek segment or land use area, are based on a common data set.

Groundwater data (unfiltered levels) were obtained from all monitoring wells along the length of EFPC. These data were aggregated and used to calculate exposure point concentrations for the risk estimates. The results of risk characterization for the agricultural and residential groundwater exposure scenarios are the same because the same exposure assumptions were used. The results for the commercial scenario differ from these two scenarios, reflecting the hypothetical use of groundwater as a source of drinking water in the work setting (Sect. 5.2).

The two principal COCs for the groundwater pathway are manganese and mercury. Using unfiltered concentrations, the HI score for manganese was as high as 28, and for mercury as high as 2.0 (based on RME assumptions). Chemicals contributing a lesser degree to the HI score include arsenic and cadmium. Unfiltered concentrations of mercury, cadmium, and lead (UCL concentrations) exceeded federal MCLs. However, concentrations of the dissolved species, (i.e., filtered samples) did not exceed MCLs.

A comparative risk assessment was conducted using filtered groundwater samples. The summed noncancer HI scores using filtered samples were lower than the unfiltered sample scores, but remained above the EPA target for noncancer effects. The reduction in observed concentrations in unfiltered versus filtered samples varied as a function of chemical. The concentration of manganese in the filtered samples did not decrease significantly, and manganese was the only substance in the filtered (dissolved) samples with an HQ greater than 1. The concentration of mercury decreased by a factor of 20 in the filtered samples, falling below the level of toxicological concern. Cancer risk estimates for the filtered samples remained above the 10^{-4} risk level due to the presence of dissolved arsenic and beryllium.

Food Chain Biotransfer. The estimates of exposure to contaminated produce (as well as for beef and dairy pathways) were not based on measured concentrations. Concentrations in home-grown produce were estimated or "modeled" using BCFs or BTFs obtained from the

literature or derived using regression equations. The BCFs are based on the notion of equilibrium partitioning between soil and plant tissue. A similar approach was adopted for the beef and dairy exposure estimates.

An experimental garden study was conducted in the EFPC floodplain to obtain site-specific data on contaminant transport into plants. Preliminary results of this study are discussed in Sect. 5.2.3. The garden study was limited in scope. With the exception of a few instances, the results did not indicate good correlation between soil levels and concentrations measured in plant tissue. The site-specific BTFs for COCs in soil were used in the Tier III risk assessment and uncertainty analysis along with published BTFs. The site-specific BTFs generally fell within the central portion of the distribution defined by the published BTF values. Examining the concentrations in plant tissue measured during the garden study, it is clear that mercury and other inorganic COCs concentrate to a much lesser extent in garden crops (one to two orders of magnitude less) than is predicted by the upper-bound BTFs obtained from the literature (i.e., NCRP publications).

Background Comparisons. Background appears to be an important component of the risk estimates, and background levels were always reflected in the risk calculations. The tolerance interval evaluation was used to determine whether concentrations at the site are statistically different from background levels at Hinds Creek. The concentrations of manganese in EFPC soils are consistent with background and are thus not site related. However, concentrations of arsenic and cadmium in EFPC soils are consistently greater than the levels observed at Hinds Creek.

Section 5.4.3.3 presented the results of risk assessment using background soil data from Hinds Creek. Although soils from Hinds Creek have not been contaminated by releases from the Y-12 Plant, high risks to human health at Hinds Creek were found, beyond EPA risk targets. Similar to EFPC, the greatest risks for the Hinds Creek assessment were associated with the food chain pathways. The risks associated with the produce pathways were very high even in the absence of any mercury contamination in Hinds Creek soils. The comparison with Hinds Creek is therefore useful in identifying which contaminants may be site related at EFPC, and which contaminants may be associated with background. Remedial action of waste sites should focus on site-related contaminants with elevated risks (i.e., exceeding EPA target ranges) above observed background levels, not for naturally occurring substances, even if they are toxic.

Uncertainty in the Risk Estimates. A great deal of uncertainty surrounds the BCFs and BTFs used in evaluating the food chain pathways. An effort has been made to characterize and

evaluate this uncertainty and its influence on the overall estimates of risk to human health. Two methods have been employed to quantitatively evaluate uncertainty:

- **Deterministic assessment**: use of two point risk estimates, an upper-bound RME estimate and a more likely or average MLE estimate, to bound the projected risks to human health.
- **Monte Carlo simulation**: probabilistic analysis of risks to human health. Risk estimates are derived in the form of probability distributions that may be statistically analyzed.

The Tier III risk assessment, presented in Sect. 5.5, is a quantitative analysis of uncertainty of the Tier II risk estimates. A detailed discussion of the uncertainty analysis follows in Sect. 5.5.

5.5 TIER III RISK ASSESSMENT AND UNCERTAINTY ANALYSIS

The results of the Tier III human health risk analysis are presented in this section. A quantitative assessment is presented of the sources of uncertainty in the exposure assessment and the relative influence on the results of risk characterization. Monte Carlo simulation has been used to develop probabilistic estimates of risk that supplement and refine the deterministic RME point estimates developed in the Tier II assessment.

The following discussion of the Tier III risk assessment includes two sections. Section 5.5.1 presents an overview of the methods of Monte Carlo simulation used to generate probabilistic estimates of risk to human health. Section 5.5.2 presents the results of the analyses.

5.5.1 Introduction

This section presents an overview of the methods of quantitative uncertainty analysis and the approach adopted for the EFPC Tier III assessment.

5.5.1.1 Overview

It is essential to recognize the uncertainty inherent in risk assessment. Uncertainty is inherent in the selection or derivation of key input parameters and in conducting every component analysis of the risk assessment process. Therefore, risk assessment of waste sites must not be viewed as yielding single value, invariant results. Rather, the results of risk assessment must be seen as estimates that span a range of possible values and that may be understood only in light of the fundamental assumptions and methods used in the evaluation.

The greatest sources of uncertainty in risk assessment are: the derivation of toxicity measures, the determination of exposure point concentrations, the development of exposure

scenarios and pathways, and the derivation of intake or dose estimates for the human receptors at greatest risk. In the traditional approach to risk assessment that has been adopted in most evaluations at National Priorities List sites, each of the variables in the risk assessment equations, including chemical concentrations, toxicity measures, and exposure parameters, is commonly taken as point estimates. In actuality, each of these variables is characterized by a distribution of possible values: a probability distribution, or more accurately, a PDF. Ideally, the BRA should generate probabilistic estimates of risk that may be statistically evaluated to quantitatively characterize uncertainty.

In the past, EPA has acknowledged the uncertainty in these point estimates and has advocated the use of conservative assumptions in the development of RME risk estimates (EPA 1989a). The intention was to err on the side of protection of human health. Following this approach, the corresponding uncertainty analysis typically would be a qualitative, order-of-magnitude evaluation or discussion of sources of uncertainty. Difficulty arises in the derivation of RME exposure estimates, as no clear, definitive guidance currently exists to present how this should be accomplished. In addition, use of conservative upper-bound point estimates for input variables results in risk projections that compound conservatism in a way that may not be meaningful or scientifically valid.

Quantitative uncertainty analysis has been incorporated into the human health risk assessment of EFPC. The objective is to develop probabilistic estimates of risk and the associated uncertainty that most meaningfully project the potential for adverse effects in exposure populations.

The current focus of the Tier III assessment is on the exposure pathways that are driving the elevated risk estimates observed in the Tier II evaluation (Sect. 5.4). The pathways included are those resulting in risks exceeding target ranges for adverse noncarcinogenic effects and excess lifetime cancer risk. The exposure pathways of principal concern are inadvertent ingestion of soil, the produce pathway, and hypothetical ingestion of groundwater.

Uncertainty will always surround estimates of exposure at waste sites. The objective is to understand, minimize, and quantify this uncertainty in risk assessment. Uncertainty in all elements of the exposure assessment are brought together and compounded in the estimate of intake or dose. It is here that the professional judgment of the risk assessor becomes particularly important. The risk assessor must examine and interpret a diversity of information, such as:

- the nature, extent, and magnitude of contamination;
- transport of chemicals in the environment;

- identification of exposure routes;
- identification of receptor groups currently at risk, and potentially at risk in the future; and
- activity patterns of receptors and receptor groups.

Based on this information, the risk assessor must develop exposure scenarios and quantify all parameters needed in the equations to estimate intake or dose (EPA 1989a).

The general form of the intake or dose equations used in risk assessment was presented and discussed in Sect. 5.2.2. The equations used will vary depending upon the exposure route under consideration (e.g., ingestion exposure and dermal exposure). For the purpose of quantifying intake or dose, each of the variables in these equations, including chemical concentration, is commonly taken as a point estimate. In actuality, each of these variables is characterized by a distribution of possible values (i.e., a PDF).

In order to quantitatively characterize uncertainty, a method is needed to propagate the uncertainty or variability in each exposure variable through to the final risk estimate. Although purely numeric methods may be used, Monte Carlo simulation is the best approach for accomplishing this given the number of variables and the complexity of the assessment. Briefly, Monte Carlo simulation is a technique for using random or pseudo-random numbers to sample from a probability distribution. The results of the sampling are used in the exposure and risk characterization equations. A distribution of possible outcomes is generated by letting a computer recalculate the risk estimates repeatedly by sampling each of the input distributions. In essence, the computer is trying to use all valid combinations of the input variables to develop (or simulate) an output distribution of risk to human health. Rather than single value results (e.g., an excess cancer risk of 2×10^{-4}), the results of risk assessment would be characterized by a distribution of possible values that could be evaluated statistically with regard to probability of exceedance (e.g., 95% probability that the cancer risk does not exceed 2×10^{-5}). Figure 5.2 depicts the use of Monte Carlo methods in risk calculation.

5.5.1.2 Uncertainty versus variability in risk assessment and Monte Carlo simulation

Before presenting the results of the Tier III risk assessment and Monte Carlo simulation, it is appropriate to discuss, in somewhat greater detail, the issue of uncertainty versus variability in risk assessment. The distinction between these two concepts was briefly introduced in the exposure assessment section of this report (i.e., Sect. 5.2.3.3, Parameter Uncertainty). This section expands upon the concepts presented in Sect. 5.2 and examines their importance in quantitative uncertainty analysis.

Standard Approach: Use of point estimates for exposure variables.

$$\text{Risk} = C \times \frac{CR \times EF \times ED}{BW \times AT} \times CSF$$

Probabilistic Approach: Use of probability distributions (PDFs) for exposure variables.

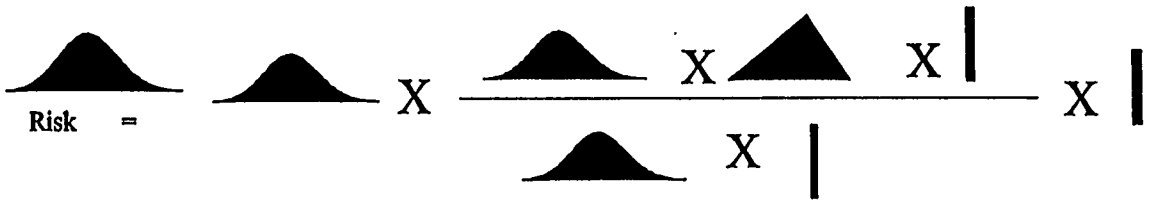


Fig. 5.2. Uncertainty Analysis Using Monte Carlo Techniques.

With regard to a given parameter whose true value is unknown, *uncertainty* describes the lack of knowledge regarding the value of the parameter, and *variability* refers to the possible differences in values for the parameter. Frey, in a recent paper prepared as part of a summer fellowship program (AAAS and EPA), emphasized the conceptual differences between uncertainty and variability in risk assessment (Frey 1992). It is his contention that these two concepts have been "muddled in traditional approaches to exposure assessment" and therefore affect the quality (reliability) of risk estimates.

In the context of exposure assessment, Frey defines variability as a heterogeneity between individual members of a population (Frey 1992). This follows Bogen's definition of variability as an abbreviated way of referring to inter-individual variability with respect to some risk-related characteristic (i.e., contact rate) that is distributed within a population at risk (Bogen 1990). In his study, Frey advocates the use of Monte Carlo analysis in human health risk assessment, but maintains that variability and uncertainty should be treated as separate distributions, describing the overall "uncertainty" for a given parameter. Thus, a two-dimensional approach to probabilistic risk assessment is proposed by Frey where the *variable* component of a given parameter is treated as a frequency distribution, and the *uncertain* component is treated as a PDF (see Sect. 5.2.3).

According to Frey, Monte Carlo simulation based on this two-dimensional approach may be used to evaluate *both* the separate and interacting effects of variability and uncertainty in the risk assessment model (e.g., risk assessment equation). Frey notes that in contrast, typical approaches to Monte Carlo simulation do not properly convey the differences between variability and uncertainty. He states that the use of "hybrid" frequency/probability distributions cannot be used as a basis for evaluating the exposure (or risk) level "faced by an individual at a given fractile of the population" (Frey 1992).

Although Frey comments that it should be possible to separate an exposure parameter into its variable and uncertain components, it is currently a formidable task to attempt such an effort for all parameters of importance in a large-scale risk assessment. The illustrative examples he provides in his study are all quite simple in comparison with the effort undertaken at EFPC (e.g., one chemical, one exposure pathway, four parameters). Further, it is unclear that the two-dimensional approach would ultimately yield results that vary significantly from the methods using the so-called hybrid probability distributions.

The uncertainty analysis conducted for EFPC does not disaggregate uncertainty and variability in the PDF developed for input parameters. However, the EFPC risk assessment team believes that the results of the Tier III assessment provide a useful measure of the uncertainty

surrounding the RME point estimates advocated by EPA. In the final analysis (as noted by Frey), it is more appropriate and defensible to make a good faith effort to characterize uncertainty, even if some or all of the efforts involve professional judgment, than to ignore the issue, presenting only the results of point estimate analyses.

5.5.1.3 Steps in conducting the Monte Carlo simulation

This section presents a step-by-step outline of the use of Monte Carlo simulation in the Tier III assessment for EFPC. The EFPC risk assessment team has attempted to be very thorough in the use of Monte Carlo methods. The sequence of analyses in the Tier III assessment includes: 1) examination of the uncertainty/variability in the input variables, 2) generation of probabilistic risk estimates and comparison with RME estimates, and 3) identification of major contributors to overall uncertainty/variability. An outline of the process is as follows:

Examine the uncertainty/variability in input variables:

- Identify or derive PDFs for each input variable that is to be treated stocastically (i.e., probabilistically).
- Run computer simulations and generate a graphical depiction of the PDF.
- Statistically evaluate the data generated by the simulation in producing the PDF for the variable: minimum, maximum, expected value, standard deviation, skewness, kurtosis, and percentile estimates.
- Examine the uncertainty surrounding the point estimate used for each exposure variable in the deterministic assessment (i.e., the Tier II creek-wide analysis). Plot the RME point estimate (for the variable) on the graphical display of the distribution (PDF). In addition, plot the 90th and 50th percentile values derived from a statistical analysis of the generated distribution.

Generate probabilistic risk estimates and examine the uncertainty/variability surrounding deterministic RME results:

- Conduct Monte Carlo simulation and examine the distribution of risk estimates for a given exposure pathway (i.e., the uncertainty surrounding the HI or cancer risk estimate). In this step, the PDFs for each uncertain variable are combined via Monte Carlo simulation to produce an output risk distribution.
- Statistically evaluate the data generated by the simulation in producing the output risk distribution (PDF): minimum, maximum, expected value, standard deviation, skewness, kurtosis, and percentile estimates.
- Plot the RME risk estimates (point estimates from the Tier II deterministic analysis) on the output risk distributions (i.e., on both probability density functions and cumulative density functions). In addition, plot the 90th and 50th percentile values derived from a statistical analysis of the distribution of risk estimates.

Identify major contributors to overall uncertainty/variability:

- Conduct a series of computer simulations to examine the contribution to the overall uncertainty in the risk estimate (i.e., for a given pathway) attributable to each exposure variable.
- This "sensitivity analysis" is accomplished by switching on and off the probabilistic treatment of each uncertain variable. Only one variable at a time is treated as a PDF. All other variables are treated as conservative point estimates.

The application of these methods and the results of the analysis are discussed below and presented in Appendix O.

5.5.2 Results of Monte Carlo Simulation

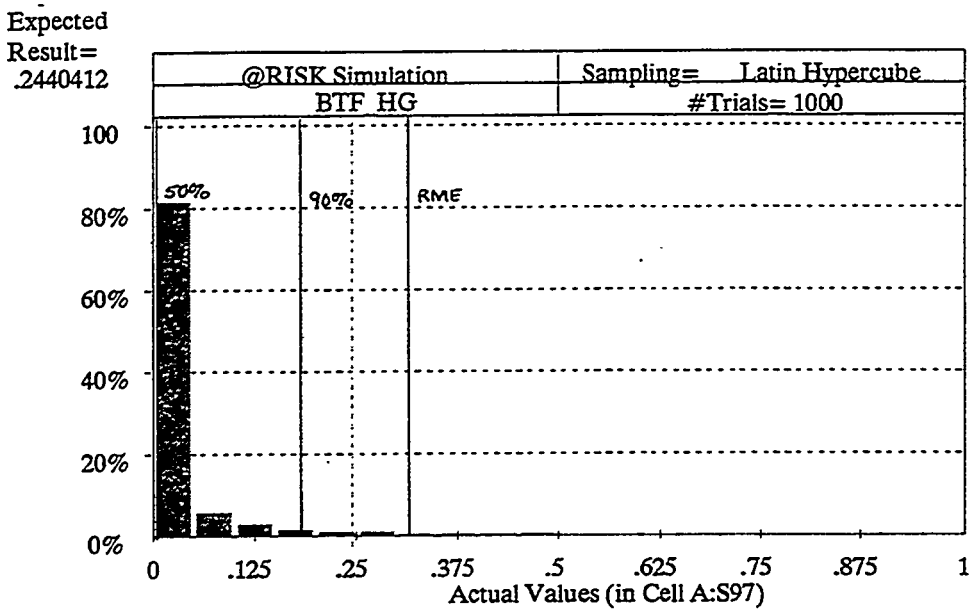
The results of Monte Carlo simulation are present in this section. Results are presented for each of the component analyses identified above in the preceding section.

5.5.2.1 Examine the uncertainty/variability in input variables

As noted above, the initial step in the quantitative uncertainty analysis is to examine the uncertainty surrounding the point estimates (i.e., RME estimates) adopted in the Tier II assessment. This is accomplished by examining the PDFs for each exposure variable in relationship to the RME point estimate. Depending upon the characteristics of the data set for a given variable, the PDF may be represented by a variety of distributions. In the assessment of EFPC, four distribution types have been used: a truncated normal, a lognormal, a truncated lognormal, and a triangular distribution. These distributions are discussed in Sect. 5.2.3 and presented in Appendix O.

In studying these PDFs, it is apparent that the RME point estimates (as would be expected) are generally quite conservative and fall in the upper tail of the probability distributions. In some cases, the RME estimates exceed the 95th percentile values. Of particular concern are the conservative point estimates used for the food chain BTFs. As an example, Fig. 5.3 presents the PDF for the BTF for mercury from soil to plant tissue. The statistical analysis of the PDF indicates that the RME point estimate exceeds the 90th percentile (i.e., there is greater than 90% probability that the BTF for mercury from soil to plant tissue is less than or equal to the RME point estimate). Recall that the point estimates used for the transfer factors were obtained from NCRP (1989, 1991). There is no compilation of transfer factors that EPA recommends for use in risk assessment, and ORNL CRAC suggested use of the NCRP values. Based upon an evaluation of the biotransfer factor PDFs for inorganic COCs, and the results of the experimental garden study (Sect. 5.2.2.10), it is concluded that these factors obtained from the literature and used in the BRA (particularly for mercury) are likely one to two orders of magnitude too high.

Fig. 5.3. East Fork Poplar Creek probability density function: soil to plant biotransfer factor – Mercury



Units:	(mg Hg/kg plant WW)/(mg Hg/kg soil)
Distribution type:	Lognormal
Mean:	−5.624
Standard Deviation:	3.004
RME/MLE Point Estimate:	0.3
Expected/Mean Result =	0.2440412
Maximum Result =	71.96641
Minimum Result =	2.757325000000E−07
Range of Possible Results =	71.96641
Chance of Positive Result =	100
Chance of Negative Result =	0
Standard Deviation =	2.537487
Skewness =	23.75243
Kurtosis =	646.4479
Variance =	6.438838
ERRs Calculated =	0
Values Filtered =	0
Simulations Executed =	1
Iterations =	1000

Percentile Probabilities
(Chance <= Shown Value)

0%	2.000000000000E−07
5%	0.00002
10%	0.00007
15%	0.0002
20%	0.0003
25%	0.0005
30%	0.0007
35%	0.0011
40%	0.0017
45%	0.0025
50%	0.0036
55%	0.0052
60%	0.0077
65%	0.0114
70%	0.0174
75%	0.0272
80%	0.0452
85%	0.0803
90%	0.1682
95%	0.4955
100%	71.9664

5.5.2.2 Examine uncertainty/variability surrounding RME estimate

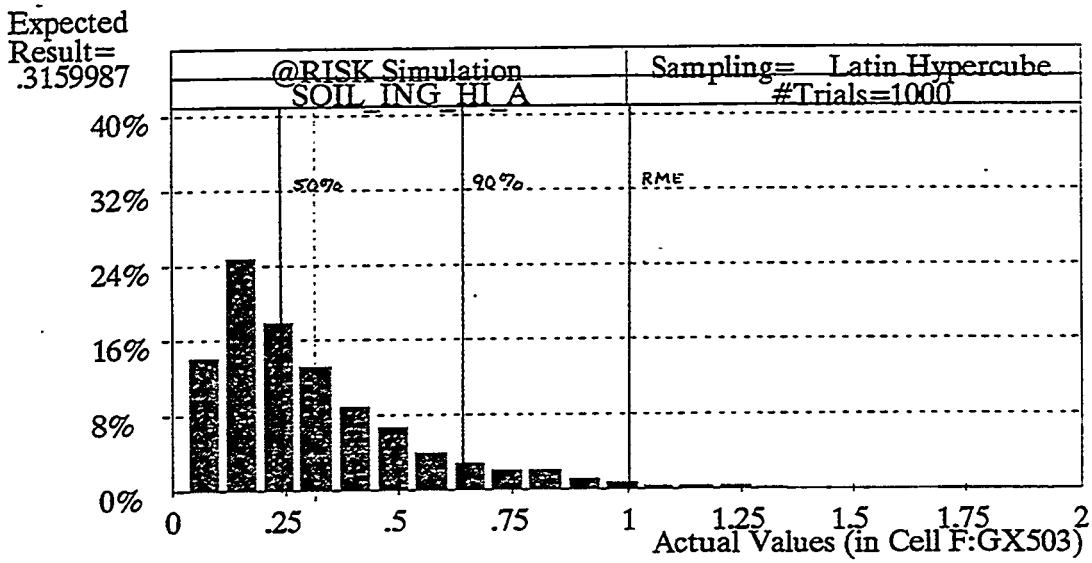
Once the PDFs for each exposure variable have been generated and evaluated, Monte Carlo simulation may be used to derive a probabilistic estimate of risks to human health. This estimate is in the form of a distribution that reflects (i.e., propagates) the uncertainty in the component input variables. Figures 5.4 through 5.9 present probabilistic risk estimates for the soil ingestion pathway. The probabilistic risk estimates for the produce and groundwater pathways are presented in Appendix O. Table 5.50 summarizes the results and presents a comparison of the RME point estimates versus the risk estimates derived from a statistical analysis of the output risk distribution. The 50th and 90th percentile estimates from the risk distribution are provided. Results are presented for adults and children, and for noncarcinogenic and carcinogenic effects.

Note that for the soil-related pathways (i.e., inadvertent ingestion and produce pathway), results have been presented only for EFPC Segment 4. An analysis of uncertainty for the soil pathways has been presented only for one segment given that the exposure assumptions for the residential and agricultural scenarios are the same across land use segments. The only difference between segments is the exposure point concentrations for the COCs. The concentrations of contaminants in soil are highest in Segment 4 (e.g., most elevated levels of mercury). Therefore, an analysis of Segment 4 will suffice as a basis for quantitatively characterizing uncertainty in the risk estimates.

The probabilistic risk estimates presented in Figs. 5.4 through 5.9 and Appendix O graphically depict the range of uncertainty in the HI scores and the incremental cancer risk estimates for the three exposure pathways under evaluation. The shape of the output risk PDF helps to establish a most likely risk estimate from within the distribution of possible values. The key point is that risk assessment does not yield single value results. Without the use of Monte Carlo simulation, there is the tendency to focus on the conservative, upper-bound risk estimates as the only possible and meaningful result. This is simply not the case.

As shown in Table 5.50, the numerical risk estimates presented in the table facilitate an evaluation of the magnitude of the range, separating the RME estimate derived through the more traditional deterministic methods from the 50th and 90th percentile estimates from the probability distribution. The 90th percentile risk estimate may be viewed as a conservative value selected from the output risk distribution (i.e., there is 90% probability that the true risk is less than or equal to the specified result). The 50th percentile estimate from the output risk distributions may be considered a best estimate of risk based on average or "most likely" exposure assumptions.

Fig. 5.4. Probabilistic risk estimate ingestion exposure of adults to soils results of Monte Carlo simulation: Hazard Index – Segment 4

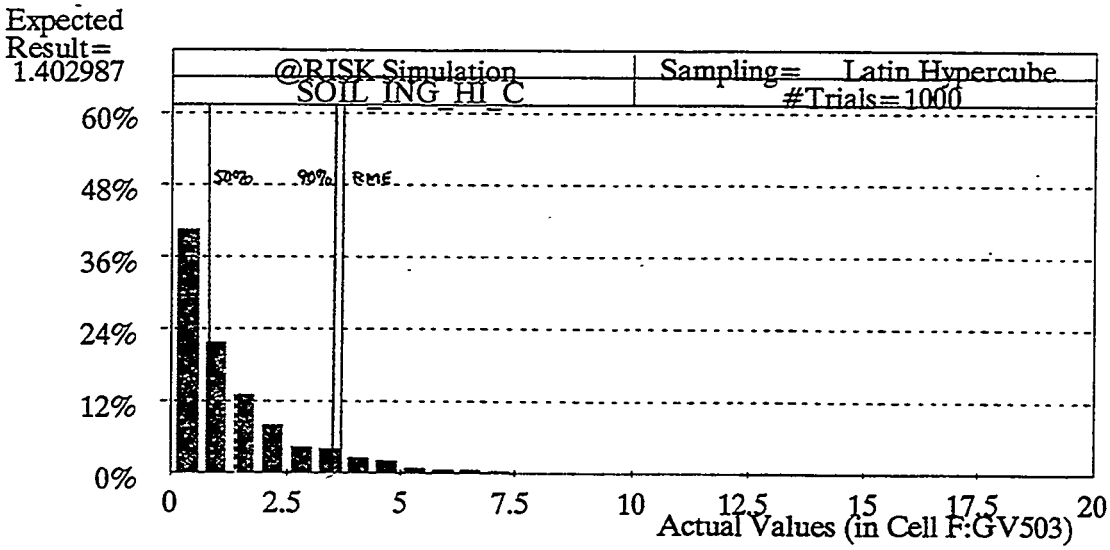


RME Point Estimate:	1.00E+00
MLE Point Estimate:	2.48E−01
Expected/Mean Result =	0.316
Maximum Result =	1.697
Minimum Result =	0.027
Range of Possible Results =	1.670
Chance of Positive Result =	100
Chance of Negative Result =	0
Standard Deviation =	0.243
Skewness =	1.873
Kurtosis =	7.715
Variance =	0.059
ERRs Calculated =	0
Values Filtered =	0
Simulations Executed =	1
Iterations =	1000

Percentile Probabilities
(Chance <= Shown Value)

0%	0.027
5%	0.074
10%	0.093
15%	0.115
20%	0.133
25%	0.147
30%	0.164
35%	0.183
40%	0.199
45%	0.225
50%	0.246
55%	0.271
60%	0.298
65%	0.329
70%	0.366
75%	0.415
80%	0.462
85%	0.522
90%	0.629
95%	0.808
100%	1.697

Fig. 5.5. Probabilistic risk estimate ingestion exposure of children to soils results of Monte Carlo simulation: Hazard Index – Segment 4

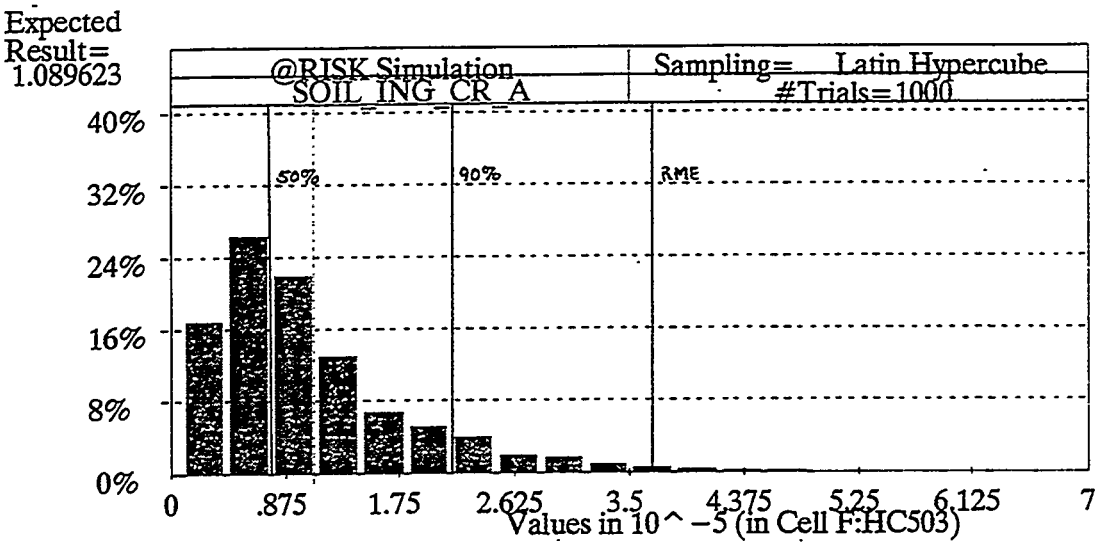


RME Point Estimate:	3.60E+00
MLE Point Estimate:	8.67E+00
Expected/Mean Result =	1.403
Maximum Result =	12.384
Minimum Result =	0.027
Range of Possible Results =	12.357
Chance of Positive Result =	100
Chance of Negative Result =	0
Standard Deviation =	1.495
Skewness =	2.269
Kurtosis =	10.734
Variance =	2.235
ERRs Calculated =	0
Values Filtered =	0
Simulations Executed =	1
Iterations =	1000

Percentile Probabilities
(Chance <= Shown Value)

0%	0.027
5%	0.138
10%	0.187
15%	0.258
20%	0.325
25%	0.391
30%	0.463
35%	0.540
40%	0.636
45%	0.743
50%	0.873
55%	0.999
60%	1.159
65%	1.418
70%	1.568
75%	1.805
80%	2.246
85%	2.723
90%	3.444
95%	4.458
100%	12.385

Fig. 5.6. Probabilistic risk estimate ingestion exposure of adults to soils results of Monte Carlo simulation: Cancer Risk (chemicals) – Segment 4

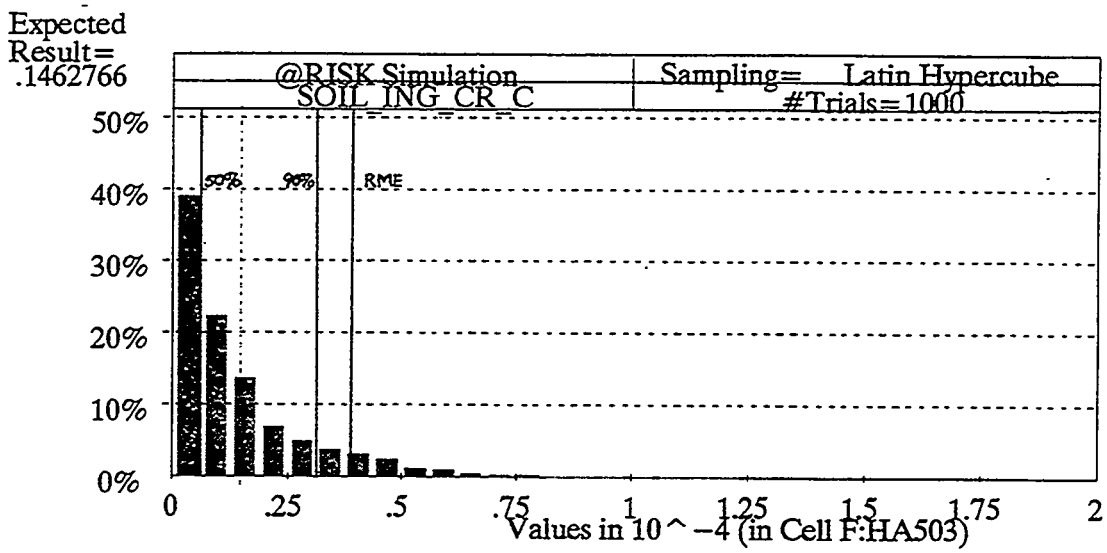


RME Point Estimate:	3.68E−05
MLE Point Estimate:	2.56E−06
Expected/Mean Result =	1.09E−05
Maximum Result =	6.93E−05
Minimum Result =	8.08E−07
Range of Possible Results =	6.85E−05
Chance of Positive Result =	100
Chance of Negative Result =	0
Standard Deviation =	8.36E−06
Skewness =	2.00E+00
Kurtosis =	8.84E+00
Variance =	6.99E−11
ERRs Calculated =	0
Values Filtered =	0
Simulations Executed =	1
Iterations =	1000

Percentile Probabilities
(Chance <= Shown Value)

0%	8.00E−07
5%	2.00E−06
10%	3.00E−06
15%	4.00E−06
20%	4.00E−06
25%	5.00E−06
30%	5.00E−06
35%	6.00E−06
40%	7.00E−06
45%	7.00E−06
50%	8.00E−06
55%	9.00E−06
60%	1.00E−05
65%	1.00E−05
70%	1.00E−05
75%	1.00E−05
80%	1.00E−05
85%	1.00E−05
90%	2.00E−05
95%	2.00E−05
100%	6.00E−05

Fig. 5.7. Probabilistic risk estimate ingestion exposure of children to soils results of Monte Carlo simulation: Cancer Risk (chemicals) – Segment 4

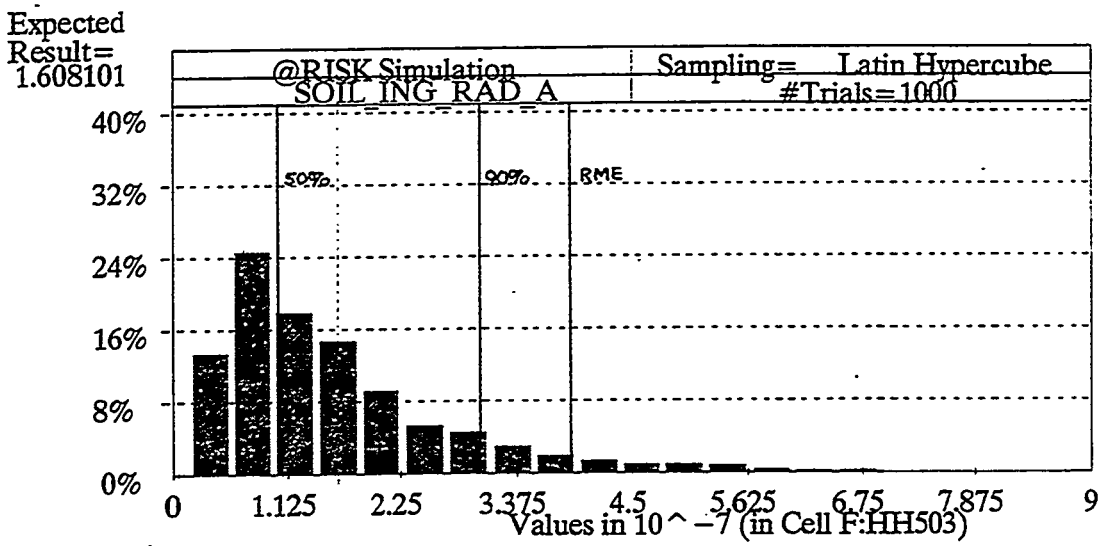


RME Point Estimate:	3.96E−05
MLE Point Estimate:	2.98E−06
Expected/Mean Result =	1.46E−05
Maximum Result =	1.24E−04
Minimum Result =	2.55E−07
Range of Possible Results =	1.23E−04
Chance of Positive Result =	100
Chance of Negative Result =	0
Standard Deviation =	1.56E−05
Skewness =	2.18E+00
Kurtosis =	9.84E+00
Variance =	2.42E−10
ERRs Calculated =	0
Values Filtered =	0
Simulations Executed =	1
Iterations =	1000

Percentile Probabilities
(Chance <= Shown Value)

0%	2.00E−07
5%	1.00E−06
10%	2.00E−06
15%	2.00E−06
20%	3.00E−06
25%	4.00E−06
30%	4.00E−06
35%	5.00E−06
40%	6.00E−06
45%	7.00E−06
50%	9.00E−06
55%	1.00E−05
60%	1.00E−05
65%	1.00E−05
70%	1.00E−05
75%	1.00E−05
80%	2.00E−05
85%	2.00E−05
90%	3.00E−05
95%	4.00E−05
100%	1.00E−04

Fig. 5.8. Probabilistic risk estimate ingestion exposure of adults to soils results of Monte Carlo simulation: Cancer Risk (radionuclides) – Segment 4

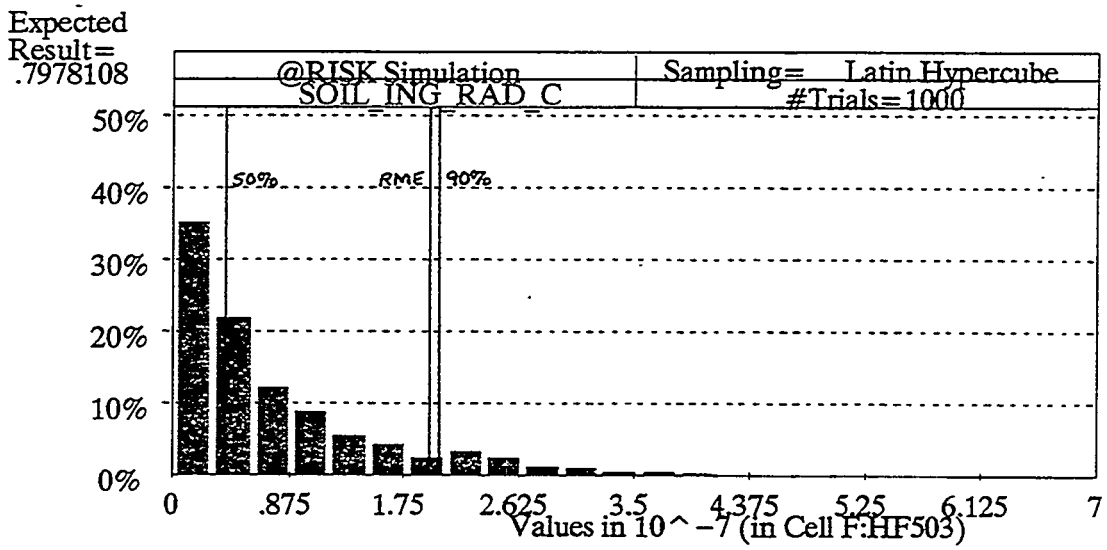


RME Point Estimate:	4.91E−07
MLE Point Estimate:	3.79E−08
Expected/Mean Result =	1.61E−07
Maximum Result =	8.62E−07
Minimum Result =	1.40E−08
Range of Possible Results =	8.48E−07
Chance of Positive Result =	100
Chance of Negative Result =	0
Standard Deviation =	1.22E−07
Skewness =	1.86E+00
Kurtosis =	7.53E+00
Variance =	1.48E−14
ERRs Calculated =	0
Values Filtered =	0
Simulations Executed =	1
Iterations =	1000

Percentile Probabilities
(Chance <= Shown Value)

0%	1.00E−08
5%	3.00E−08
10%	4.00E−08
15%	6.00E−08
20%	6.00E−08
25%	7.00E−08
30%	8.00E−08
35%	9.00E−08
40%	1.00E−07
45%	1.00E−07
50%	1.00E−07
55%	1.00E−07
60%	1.00E−07
65%	1.00E−07
70%	1.00E−07
75%	2.00E−07
80%	2.00E−07
85%	2.00E−07
90%	3.00E−07
95%	4.00E−07
100%	8.00E−07

Fig. 5.9. Probabilistic risk estimate ingestion exposure of children to soils results of Monte Carlo simulation: Cancer Risk (radionuclides) – Segment 4



RME Point Estimate:	1.96E−07
MLE Point Estimate:	1.58E−08
Expected/Mean Result =	7.98E−08
Maximum Result =	5.89E−07
Minimum Result =	1.30E−09
Range of Possible Results =	5.87E−07
Chance of Positive Result =	100
Chance of Negative Result =	0
Standard Deviation =	8.24E−08
Skewness =	1.89E+00
Kurtosis =	7.27E+00
Variance =	6.79E−15
ERRs Calculated =	0
Values Filtered =	0
Simulations Executed =	1
Iterations =	1000

Percentile Probabilities
(Chance <= Shown Value)

0%	1.00E−09
5%	8.00E−09
10%	1.00E−08
15%	1.00E−08
20%	1.00E−08
25%	2.00E−08
30%	2.00E−08
35%	3.00E−08
40%	3.00E−08
45%	4.00E−08
50%	4.00E−08
55%	5.00E−08
60%	6.00E−08
65%	7.00E−08
70%	9.00E−08
75%	1.00E−07
80%	1.00E−07
85%	1.00E−07
90%	2.00E−07
95%	2.00E−07
100%	5.00E−07

Table 5.50. Comparison of RME estimates with results of Monte Carlo analysis for East Fork Poplar Creek Segment 4 (future land use – residential)

Pathway	Children			Adults		
	RME	90%	50%	RME	90%	50%
Soil Ingestion						
Noncarcinogenic Effects						
Hazard Index	3.6E+00	3.4E+00	8.7E−01	1.0E+00	6.3E−01	2.5E−01
Carcinogenic Effects						
Risks to Chemicals	4E−05	4E−05	9E−06	4E−05	2E−05	9E−06
Risks to Radionuclides	2E−07	2E−07	5E−08	5E−07	3E−07	1E−07
Groundwater Ingestion[a]						
Noncarcinogenic Effects						
Hazard Index	3.4E+01	3.9E+01	1.7E+01	2.9E+01	2.0E+01	1.0E+01
Carcinogenic Effects						
Risks to Chemicals	1E−04	1E−04	6E−05	4E−04	2E−04	1E−04
Risks to Radionuclides	3E−06	3E−06	1E−06	2E−05	2E−05	7E−06
Produce Ingestion[b]						
Noncarcinogenic Effects						
Hazard Index	3.1E+02	6.2E+01	4.4E+00	1.5E+02	2.6E+01	1.8E+00
Carcinogenic Effects						
Risks to Chemicals	3E−04	1E−04	3E−05	5E−04	2E−04	4E−05

[a] Risk assessment results shown here are based on unfiltered samples characterized by high concentrations of suspended material. A supplemental risk analysis was conducted using filtered groundwater samples as as a point of comparison (see Sect. 5.4).

[b] Note: the soil to plant biotransfer PDFs obtained from the literature were originally expressed on a dry weight basis. A conversion factor of 15.02 (grams dry−weight tissue/grams wet−weight tissue) was used to convert these factors to wet weight basis. Risk estimates include chemicals for which PDFs are available (including the RME estimate). Other chemicals and radionuclides were excluded from the derivation of probabilistic risk estimates (see Section 5.2 for details).

As Table 5.50 shows, the RME point estimates in general are approximately 4 to 100 times greater than the 50th percentile estimates. The greatest variation between the RME estimate and the 50th percentile estimates from the Monte Carlo simulation are for the food chain (i.e., produce) pathway (Table 5.50). It is illustrative to compare the results of the deterministic assessment and the risk estimates from the probability distributions. PDFs for soil-to-plant biotransfer are not available for all subject contaminants in soils.

The HI score (RME estimate, produce pathway) for adults based on the deterministic assessment is approximately 1.5×10^{2}. This value is approximately a factor of 100 times higher than the 50th percentile estimates obtained from probability distributions generated in the Tier III probabilistic analysis (HI = 1.78). It is approximately an order of magnitude higher than the 90th percentile estimate. Results for the cancer risk estimates are characterized by less uncertainty (i.e., attributable to the exposure assumptions) than the estimates for noncarcinogenic effects. The results for exposure of children to produce show the same trend.

The difference between the RME point estimate for the groundwater ingestion pathway and the 90th and 50th percentile estimates from the probability distributions is much less pronounced than for the food chain pathways. The difference between the RME estimate and the 50th percentile value is approximately a factor of 2 to 3. This reflects risk estimates characterized by much less uncertainty/variability than that for the food chain pathways. Note that the results for the groundwater pathway presented in Table 5.50 are based on unfiltered groundwater samples. These results indicate risks that exceed the target ranges established by EPA for waste site remediation under the Superfund program.

As discussed in Sect. 5.4, risk assessment for the groundwater pathway also has been conducted using filtered samples as a point of comparison. Although Monte Carlo simulation was not conducted using the filtered samples, based on the RME point estimate, the 50th percentile of the probability distribution for the HI score is likely to be less than 1.0 and the cancer risk estimate less than 10^{-4}.

Similar to groundwater ingestion, the results of the Monte Carlo simulation for the soil ingestion pathway show much less variation (i.e., than the produce pathway) between the RME estimates and the 90th percentile and 50th percentile results from the output risk distribution. The difference between the RME estimate and the 50th percentile value is approximately a factor of 4.

5.5.2.3 Identify major contributors to overall uncertainty/variability

In order to investigate the degree to which each uncertain exposure variable contributes to the overall uncertainty in the risk estimate for the exposure pathway, a final series of Monte Carlo simulations was conducted. The spreadsheet model used in the simulation was built incorporating a software switch or toggle that allows the operator of the program to turn on or off the probabilistic treatment of the exposure variables. That is, each exposure variable could be treated either as a point estimate or as a PDF.

In this analysis, the Monte Carlo simulation was run repeatedly and the output probabilistic risk distribution was generated. In the first run, all uncertain exposure variables where treated as PDFs. The results of this analysis have been presented above. In subsequent runs, however, only one of the exposure variables at a time was treated as a distribution, and all others were held as RME point estimates. In this way, the influence of the uncertainty in each of the variables may be evaluated with respect to the overall uncertainty in the risk estimate. In the assessment of EFPC, this evaluation is referred to as a "multivariable comparative analysis of uncertainty." It may be viewed as a form of sensitivity analysis using Monte Carlo methods.

Note that regression analysis also may be used to examine the extent to which each uncertain exposure variable contributes to the overall uncertainty in the risk estimate. Standardized regression coefficients (SRCs) may be used to quantify the relative contribution of the uncertainty in the input variables on the uncertainty of the output variables. Frey (1992) briefly discusses this method in his recent paper. In this technique, all sample values for the model input variables are standardized and a multi-variate regression is conducted: output variate based on the inputs. The regression coefficients for the input variates may then be used to examine the relative importance of this input parameter in determining the output values. SRC analysis is commonly limited to cases where the relationship between input and output variables is linear. It is considerably more difficult to extend this technique to nonlinear cases.

The simulation technique adopted in the study of EFPC is a viable alternative method to SRC analysis and is more easily accomplished once the spreadsheet model has been developed. Figure 5.10 is an example output of the multivariable comparative analysis (soil ingestion exposure to adult, noncancer effects). The output from these analyses is presented in two forms: as PDFs and cumulative density functions (CDFs). Results for the remainder of the soil pathways, as well as for groundwater and produce ingestion, are presented in Appendix O.

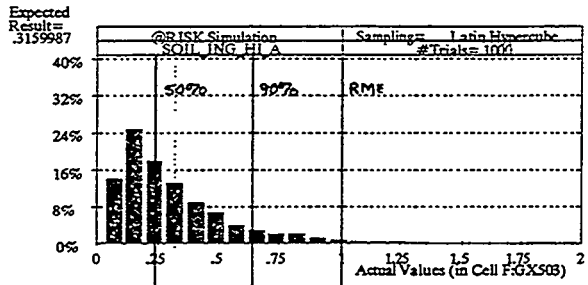
Before examining these results, it is helpful to review the differences between the CDF and the PDF described previously. The CDF depicts the cumulative probability density moving

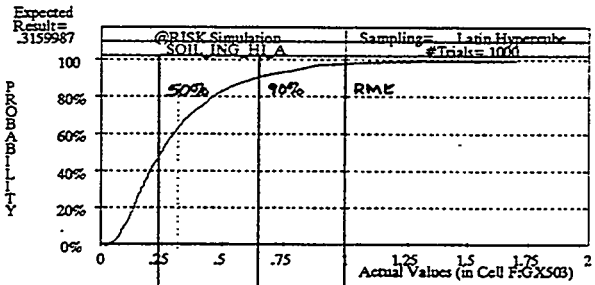
Fig. 5.10. Multivariable comparative analysis of uncertainty – ingestion exposure of adults to soils results of Monte Carlo simulation: Hazard Index – Segment 4

Soil ingestion (residential land use)
Hazard Index: soil ingestion – adult

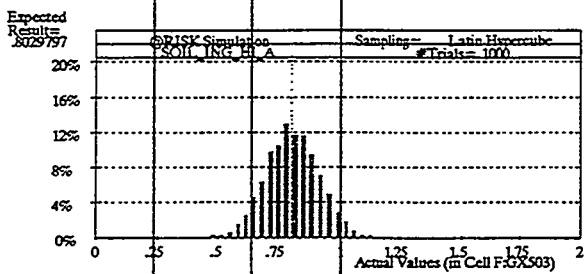
Soil ingestion (residential land use)
Hazard Index: soil ingestion – adult

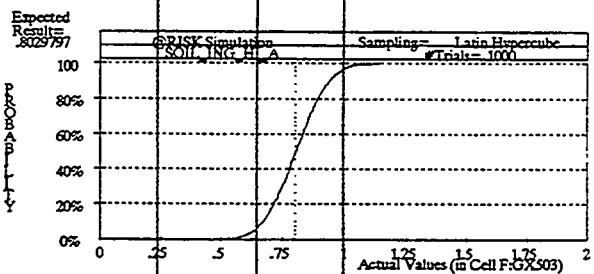
ALL TOGGLES ON



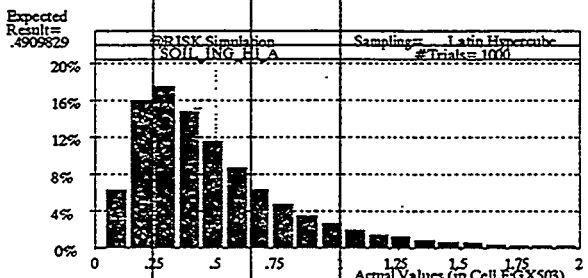


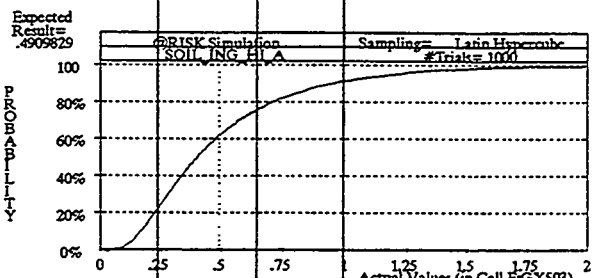
EXPOSURE POINT CONCENTRATION TOGGLE ON



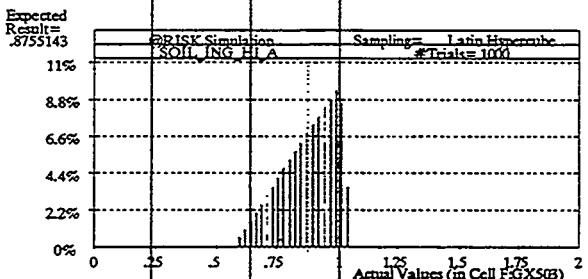


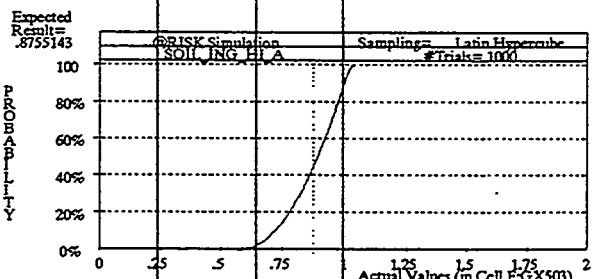
CONTACT RATE TOGGLE ON



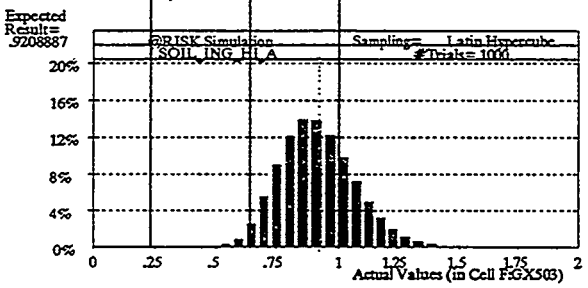


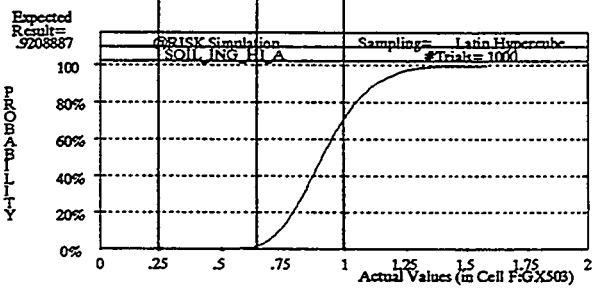
EXPOSURE FREQUENCY TOGGLE ON





BODY WEIGHT TOGGLE ON





across the range of risk estimates presented in the PDF. The CDF can be thought of as the integral of the PDF (i.e., it integrates the area under the PDF curve). The PDF is useful in examining the shape of the probability distribution and in identifying most likely (i.e., probable) values. The CDF, on the other hand, facilitates the calculation of confidence intervals and the comparison of uncertain variables. For a given risk estimate along the X axis, the PDF shows the relative probability of obtaining this particular value (e.g., 20% probability that the cancer risk is *equal* to 2×10^{-4}), whereas the CDF indicates the cumulative probability that the risk is less than or equal to this value (e.g., 95% probability that the risk is *less than or equal to* 2×10^{-4}). Identifying the principal sources of uncertainty in the final risk estimates is made easier by the simultaneous presentation and comparison of PDFs and CDFs.

In Fig. 5.10, the first set of distributions shown (i.e., labeled "all toggles on" in the top row) present the probabilistic distribution of possible HI scores generated with all input variables treated stocastically. The X axis of all of the distributions shows the value of the HI score for the soil ingestion pathway. Overall, the figure shows that the principal source of uncertainty is the variation in contact rate. Note the similarity in the shape of the PDFs for contact rate and the output risk distribution (i.e., at the top of the column).

Second, note that the CDF for contact rate accounts for the greatest proportion of the cumulative risk density and any of the variables examined (i.e., the greatest contributor to the cumulative density of the output risk distribution). Similar results are observed for the assessment of carcinogenic effects in adults (Appendix O).

Appendix O presents the remaining results of the multivariable comparative analysis for soils. For children (ages 3 to 12 years), contact rate and body weight are the principal contributors to the overall uncertainty in the HI score. Looking at the CDFs for these two variables, body weight is actually stocastically dominant. This implies that a smaller percentage of the overall uncertainty in the RME estimate may be attributed to the variation in body weight than to the uncertainty in contact rate.

The results of the multivariable comparative analysis for the groundwater and produce pathways also are presented in Appendix O. The RME point estimates are very conservative and exceed the 95th percentile estimate of the risk distribution. Similar to the results for the soil ingestion pathway, contact rate and body weight contribute most greatly to the overall uncertainty in the probabilistic risks estimates (both the HI score and the excess lifetime cancer risk estimates).

5.5.3 Implications of Results of Monte Carlo Simulation

The Tier III assessment using Monte Carlo simulation is an attempt to develop estimates that most meaningfully characterize the potential risks to human health. In understanding this effort, it is important to reiterate a fundamental issue previously discussed in the report:

> *In human health risk assessment, as in all evaluations involving uncertain quantities, there is no single correct answer.*

Risk assessment does not yield single value results because *none of the input parameters to the risk assessment equations and models is known with absolute certainty*. Acknowledging the limitations of our understanding, risk assessors must thus strive to achieve a balance between the desire to *ensure* protection of human health and the environment (i.e., through the introduction of highly conservative assumptions), and the need to develop sound, scientifically defensible, estimates of risk.

In light of uncertainty in risk assessment, there has been an understandable tendency to err on the side of protection. The approach has been one of building sufficient conservatism into most elements of risk quantification so as to be confident that there is only a small likelihood that risks to human health have been underestimated. This conservatism has its place in preliminary, screening-level analyses used to explore the potential for adverse effects in exposed populations (e.g., Tier I analysis of EFPC). If the results of such analyses fall within the target ranges established by federal and state regulatory agencies, risk assessment need not progress beyond this preliminary phase. However, if these conservative estimates exceed the target ranges, the basic assumptions should be examined and the risk assessment re-evaluated.

EPA attempted to address this issue by developing the concept of RME. Rather than a highly conservative worst-case evaluation, the RME evaluation was designed to be a compromise. As designed, the RME estimate would afford adequate protection of human health without developing overly conservative, unrealistic exposure and risk estimates. However, this approach still remains problematic because:

- There is no clear cut, uniform approach for defining the RME estimate. For example, EPA specifies that the RME estimate should be derived by combining conservative (e.g., 95%) and average (e.g., 50%) values for exposure variables. Although guidance is available on default exposure parameters, it is much less clear as to how to achieve this mix of 95% and 50% values.
- No matter how the RME estimate is derived, it remains a single point estimate. As a single number, this risk estimate in no way reveals how much conservatism and uncertainty/variability is built into the results. A single point estimate presented as the

result of risk assessment introduces a false sense of precision. It masks how little is known about the true value of the exposure variables and toxicity measures that form the basis of the risk estimates.

From these considerations we recognize the need for a more rigorous approach to dealing with uncertainty. Monte Carlo simulation is a useful technique in this regard.

Monte Carlo simulation allows the risk assessor to treat each input variable as a probability distribution rather than a single point estimate. Using computer simulation, a true probabilistic estimate of risk may be developed. In the baseline human health risk assessment of EFPC, Monte Carlo simulation has allowed the EFPC risk assessment team to quantify the uncertainty/variability surrounding the exposure assumptions and the RME point estimates of risk. Further, the output risk distributions may be statistically evaluated to examine the probability that a given risk estimate is less that or equal to any given value.

Throughout this study, the EFPC risk assessment team has attempted to be thorough and clear in describing the methods of Monte Carlo simulation. However, the techniques and theory of quantitative uncertainty analysis considerably extend beyond that required in deterministic risk assessments. Further, EFPC is one of the largest study areas for which the methods of Monte Carlo analysis have been applied in human health risk assessment. A large body of information has been provided in this report and it is easy to lose sight of the fundamental objects of the Tier III analysis:

- explore the uncertainty/variability surrounding the RME estimates for exposure variables,
- quantify the uncertainty/variability surrounding the RME estimates of risks to human health, and
- interpret results in light of EPA target (i.e., benchmark) risk levels for cancer and noncancer effects.

In the EFPC BRA, Monte Carlo simulation has *not* been used as a replacement for EPA methods outlined in RAGS (EPA 1989a). As noted previously, Monte Carlo simulation has been used to supplement the conservative, deterministic assessment (i.e., Tier II analysis) based on RME estimates. The ultimate benefit of the quantitative uncertainty analysis may be realized in the feasibility study where risk management and the selection of remedial alternatives become key issues. The information derived from the Tier III analysis will facilitate decisionmaking by assisting in balancing costs, technical feasibility, and potential risks to human health.

Figures 5.11 and 5.12 graphically depict one approach for interpreting the results of Monte Carlo simulation in light of the results of the deterministic (Tier II) risk assessment based on

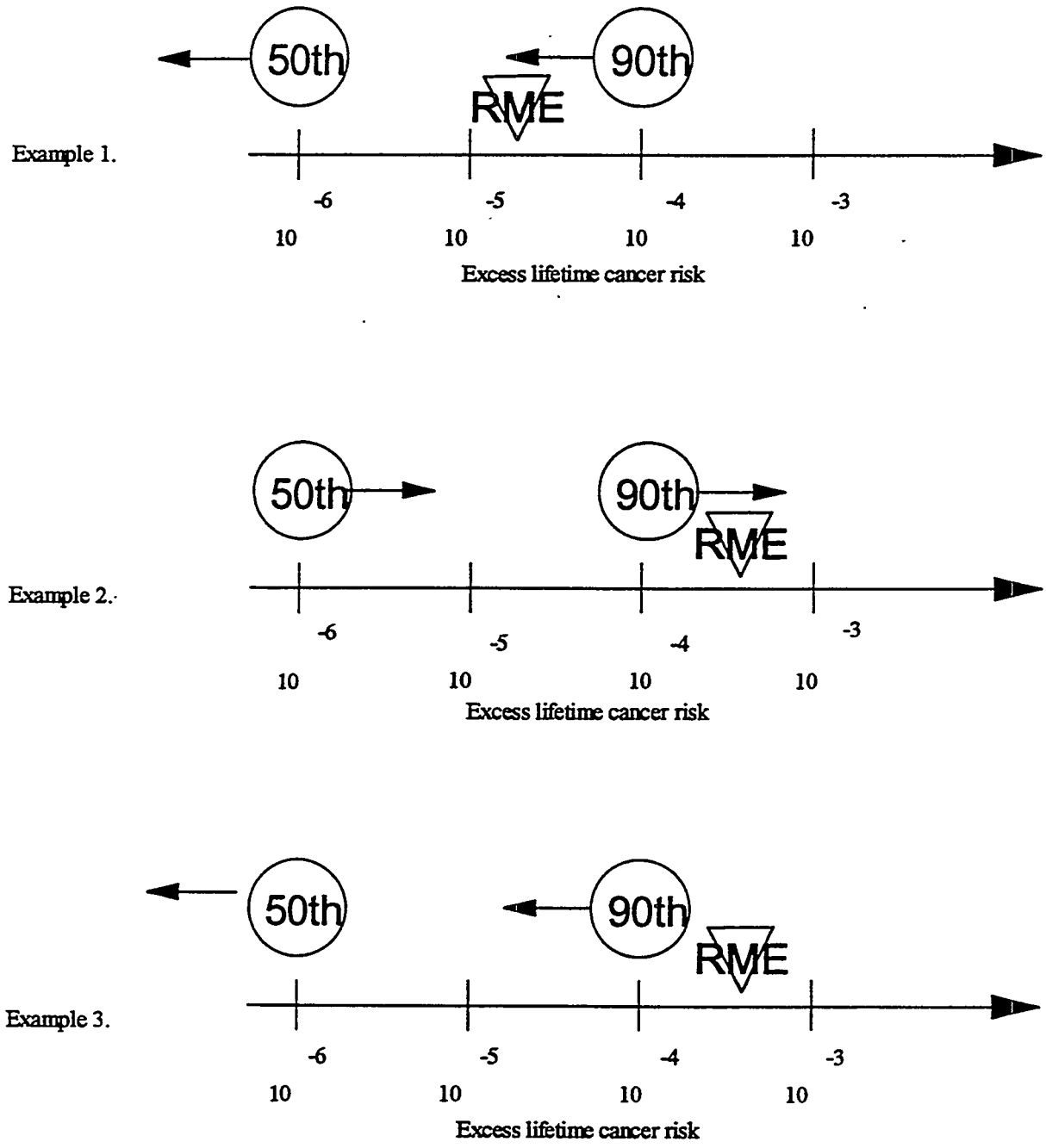
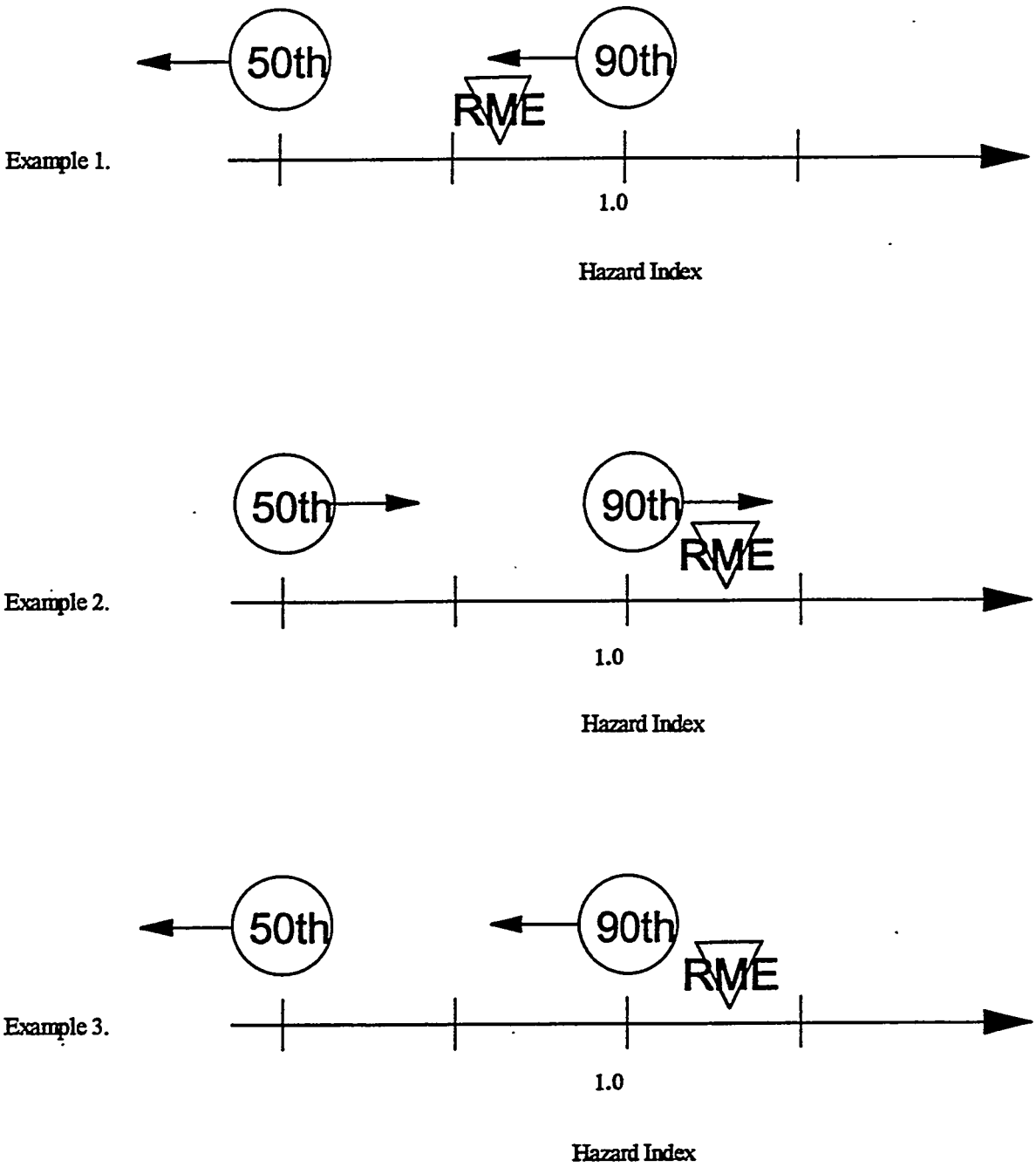


Fig. 5.11. Interpreting the results of Monte Carlo simulation: cancer risk estimates.



= Probabilistic risk estimates as percentiles from output PDF: Tier III assessment.

RME = Reasonable maximum exposure risk estimates: Tier II deterministic assessment.

Fig. 5.12. Interpreting the results of Monte Carlo simulation: noncancer risk estimates.

RME assumptions. The approach shown is an extension of ideas first proposed by Dr. David Burmaster, president of Alceon Corporation, who supported the EFPC risk assessment effort. In Fig. 5.11, the RME estimate of excess lifetime cancer risk is compared with probabilistic estimates derived from Monte Carlo simulation. Recall that the output from the Monte Carlo simulation is a probabilistic distribution of risk estimates that may be statistically analyzed. In the human health evaluation of EFPC, 90% and 50% risk estimates are identified from the output risk distributions. Similarly, Fig. 5.12 depicts an approach for interpreting the noncancer risk estimates.

Three example outcomes are shown in Figs. 5.11 and 5.12:

- **Example 1:** The RME point estimate falls within the EPA target range. The 90% estimate derived from the Monte Carlo simulation also fall within this range and thereby supports the conclusion of the Tier II (deterministic) analysis. These results would support a decision of no action at the site under investigation.
- **Example 2:** The RME estimate exceeds the target risk range established by EPA, as does the 90% value from the Monte Carlo simulation. The uncertainty analysis supports the conclusion of the Tier II assessment. These results would support a conclusion of the need for site remediation or further investigation.
- **Example 3:** The RME estimate exceeds the target risk range established by EPA. However, the 90% estimate from the Monte Carlo simulation falls *within* the EPA target risk range. These results may be indicative of an extremely conservative point estimate (i.e., Tier II analysis) and point to the need for further refinement and evaluation of the risk assessment.

Monte Carlo simulation was conducted using monitoring data and the exposure assumptions for Segment 4 of the EFPC floodplain. The highest levels of contamination and the most conservative exposure assumptions were used in the assessment of this area. Table 5.50 presented the comparative results of the RME (i.e., Tier II) versus 90% and 50% risk estimates (i.e., Tier III) for children and adults. Results are presented for the three exposure pathways found to drive the risk assessment (i.e., results of the Tier II deterministic analysis).

As shown in Table 5.50 (quantitative uncertainty analysis for Segment 4), the results may be described by Example 1 or Example 2 above in all but one case. That is, the Monte Carlo simulation supports the conclusions of the deterministic assessment. The exception was the soil ingestion pathway for adults. Here the RME HI estimate was approximately 1.0. The Monte Carlo simulation indicates with greater than 90% probability that the results fall within the target risk range established by EPA.

The approach described above (i.e., the three "examples") is a first attempt at establishing a convention for interpreting the results of Monte Carlo simulation in human health risk assessment. Although the powerful techniques of quantitative uncertainty analysis provide additional insight into the validity of the risk estimates, these results should not stand alone. The results of risk characterization must be interpreted in light of the fundamental assumptions that form the basis of the exposure and toxicity assessments. For example, it is necessary to revisit the assumptions regarding current and future land use designation, receptors at risk, exposure point concentrations and pathways, derivation of intake and dose estimates, and stability of the EPA toxicity measures used in the study. In the final analysis, many lines of reasoning must be brought together to put into perspective the more quantitative aspects of the risk assessment process.

By way of conclusion, Monte Carlo simulation must be recognized as an evolving tool in human health risk assessment. As such, a number of challenges must be faced in enhancing the utility of this technique:

- distinguishing between uncertainty and variability in risk assessment;
- identifying the variables that should be treated stocastically, and those that should be treated deterministically;
- Identifying PDFs for each uncertain variable (type, shape) and building a library of PDFs for future use in risk assessment;
- determining the defining parameters for the PDFs: mean, standard deviation, and minimum, maximum, and most likely values; and
- identifying and accounting for correlation between variables.

Although all tools are not yet available to use Monte Carlo simulation as the sole basis for a large-scale human health risk assessment, the technique remains a valuable adjunct to the more traditional methods established by EPA. The methods of probabilistic risk assessment make explicit the uncertainty/variability and conservatism in risk estimates based upon RME assumptions. The simultaneous presentation of the results of both deterministic and probabilistic risk assessments provides the maximum amount of information to risk managers and the regulatory community in evaluating the need for site remediation.

5.6 CONCLUSIONS: BASELINE HUMAN HEALTH RISK ASSESSMENT

The conclusions for the baseline human health risk assessment at EFPC are presented in this section. The discussion summarizes the results of the deterministic and probabilistic assessments. Five subsections are presented: general conclusions for the study; conclusions for the soil ingestion pathway; conclusions for the food chain pathway; conclusions for the groundwater

pathway; and conclusions regarding the toxicity of mercury and its influence on the human health risk estimates.

5.6.1 General Conclusions

No imminent or substantial endangerment to human health is associated with exposure to contaminants in environmental or biological media at EFPC. The results of the BRA do not indicate the need for immediate short-term action to mitigate potential exposures or risks to human health.

Risk assessment based on the use of RME assumptions (i.e., the Tier II assessment using the traditional deterministic methods) yields very conservative results and indicates the potential for adverse noncarcinogenic and carcinogenic effects associated with long-term exposure to contaminants in EFPC. Risk estimates exceeded the target ranges established by EPA under the Superfund program for waste site remediation. Mercury was confirmed as the principal COC in EFPC floodplain soils.

For noncarcinogenic effects, risk estimates were designated of concern (i.e., exceeding the target range) if the HQ for any given contaminant or the HI for combined exposure across contaminants exceeded a value of 1. Estimates of excess lifetime cancer risk to the individual that exceeded 1×10^{-4} were designated of concern. (Excess lifetime cancer risk estimates falling within the range of 10^{-6} to 10^{-4} *may* be designated as acceptable by EPA. The acceptability of risk results in this range is determined by the Regional Project Manager and may depend upon: the circumstances surrounding exposure to receptor populations, the ability to detect the COCs, the projected environmental fate of the COCs, and the uncertainty surrounding the toxicity measures and final risk estimates).

The Tier III risk assessment and quantitative uncertainty analysis demonstrates the conservatism associated with the RME estimates. Monte Carlo simulation was used to make explicit the range of uncertainty/variability associated with the point estimates of risk to human health.

Four land use areas were established as the basis of the human health risk assessment: agricultural/homesteader, residential, open land use (hiking or trespassing), and commercial land use. Both current and future land use scenarios were considered in the BRA. Risk assessment for combined exposure across pathways for the residential and agricultural scenarios always resulted in unacceptable risks to human health. This is true under both current and future land use assumptions. The open land use scenarios never resulted in unacceptable risks to human

health for either current or future land uses. The commercial land use scenario was based on hypothetical future use of groundwater. These results exceed the target range established by EPA.

Risks exceeding EPA target ranges were associated with exposure to chemical contaminants. The risks associated with exposure to radionuclides fell within the target range established by EPA in all cases.

The exposure pathways of primary concern for the residential and agricultural scenarios are: inadvertent ingestion exposure to soils; the food chain pathways that examine the movement of contaminants from soil to produce, beef, and dairy products; and hypothetical use of groundwater as a source of drinking water. The results of risk assessment of recreational use of EFPC (i.e., use of the creek for fishing, swimming, and wading, including dermal contact with sediment) fell within the target range established by EPA.

5.6.2 Conclusions: Inadvertent Ingestion Exposure to Soil

RME estimates of risks of adverse noncarcinogenic effects associated with inadvertent ingestion exposure to soils exceeded the target range established by EPA in Segments 1, 3, 4, 5, and 8. Children were determined to be at higher risk than adults. Cancer risk estimates for soil ingestion are primarily associated with the presence of arsenic and beryllium. The risk estimates for all segments fell within the target range established by EPA for waste site remediation.

Based on the evaluation of inadvertent exposure to soil, mercury was determined to be the principal COC. This was true in all cases where the HI score exceeded 1. Chemicals contributing a lesser degree to the HI score include cadmium, arsenic, and manganese.

Concentrations of contaminants in floodplain soils were compared with background concentrations at the Hinds Creek reference site using a tolerance interval approach. Manganese and beryllium were present in EFPC soils at levels comparable to (indistinguishable from) background levels. Concentrations of mercury, cadmium, and arsenic were substantially above observed background levels at Hinds Creek.

The Monte Carlo analysis of the soil ingestion pathway (Tier III assessment) evaluated the range of potential risk estimates in light of the uncertainty surrounding the exposure variables. The RME point estimates for adults exceeds the 90th percentile of the probabilistic distribution of risk estimates. The point estimate is approximately four times greater than the 50th percentile risk estimate derived from the Monte Carlo simulation. The Tier III assessment demonstrates that

less than an order of magnitude uncertainty/variability (i.e., associated with the exposure assumptions) exists in the RME point estimates.

5.6.3 Conclusions: Food Chain Pathways

Very limited agricultural activity currently occurs along EFPC. A few property owners raise horses and beef cattle. However, no dairy cattle are being raised on floodplain soils and large-scale farming of crops does not exist. A few gardens are located within the vicinity of floodplain soils, but active gardening is limited in the EFPC floodplain as a whole. In essence, the analysis of the food chain pathways is an assessment of future land use potential and an effort to comprehensively evaluate potential risks to human health.

RME risks estimates associated with the food chain pathways greatly exceed the targets established by EPA for waste site remediation. This is true for all segments and land use areas for which residential and agricultural scenarios are the basis of the risk analysis. The pathways of concern (i.e., resulting in elevated risks) are ingestion of produce, beef, and dairy products. The produce pathway accounted for the greatest percentage of the risk to human health. Ingestion of fish did not result in unacceptable risks to human health.

The extremely high risk estimates for the produce pathway are principally associated with the presence of four contaminants. In order of overall contribution to the estimate of adverse noncarcinogenic effects, the chemicals are mercury, manganese, cadmium, and arsenic. Arsenic was the basis of the unacceptably high excess lifetime cancer risk estimate. Levels of manganese in EFPC cannot be distinguished from background concentrations at Hinds Creek. As noted above, concentrations of mercury, arsenic, and cadmium consistently exceed observed concentrations in background soils.

The estimates of exposure to contaminated produce (as well as for beef and dairy pathways) were not based on measured concentrations. Concentrations in home-grown produce (as well as beef and dairy) were estimated or "modeled" using BCFs or BTFs obtained from the literature (or derived using regression equations) and assuming equilibrium partitioning between soil and plant tissue. In order to further explore the significance of the risk estimates and the uncertainty surrounding the results, a series of additional analyses were conducted:

- An experimental vegetation and garden plot study was conducted to empirically evaluate concentrations of contaminants in plant tissue.
- Risk assessment was conducted using the background data for soils from Hinds Creek. Exposure assumptions for the agricultural scenario were the basis of this assessment.

- Monte Carlo simulation was conducted (Tier III risk assessment) to evaluate the uncertainty associated with the RME point estimates of risk to human health.

The conclusions from these supplemental analyses are presented below.

The experimental vegetation and garden plot study indicated that mercury and other floodplain soil contaminants bioconcentrate in plant tissue to a much *lesser* extent than that predicted using food chain modeling. Although a significant and meaningful correlation between soil and plant tissue levels could not be established from the experimental data, the results of plant tissue analysis were useful and demonstrated limited soil-to-plant transfer and concentration of contaminants in plant tissue.

The risk assessment using background soil data from Hinds Creek resulted in estimates of risk to human health for the food chain pathways that greatly exceed the target range established by EPA for waste site remediation. The risks associated with direct soil contact fell within the acceptable range established by EPA. Even though the Hinds Creek study area has not been affected by discharges from the Y-12 Plant (e.g., there is no mercury contamination of soils), the risks of hypothetical food chain exposure to background concentrations of chemicals in soils were very high. This reflects the conservative assumptions used in the analysis of the food chain pathways. In particular, it reflects conservatism in the BTFs used to model chemical movement from soils to plant, beef, and dairy.

Monte Carlo simulation was used to derive probabilistic estimates of risk to human health associated with the food chain pathways. Risk estimates derived from the Monte Carlo simulation (Tier III analysis) were compared with the RME risk assessment from the Tier II study. The RME (point) risk estimate for noncancer effects for the produce pathway exceeds the 90th percentile risk estimate derived from the probability distribution. The RME estimate is a factor of 100 times higher than the 50th percentile (i.e., most likely or average) estimate from the probability distribution.

In summary, a great deal of uncertainty surrounds the estimates of risk to human health associated with the food chain pathways. Given the conservatism built into the analysis, and the fact that very limited agricultural activity is occurring along EFPC (or is likely to occur in the future), it is concluded that the *RME risk estimates* for the food chain pathways are not a useful decisionmaking tool. The results of the Tier III Monte Carlo simulation indicate that additional evaluation of site-specific biotransfer into the food chain is required if the food chain pathways are to be used as a basis for determining the need for site remediation.

5.6.4 Conclusions: Hypothetical Ingestion Exposure to Groundwater

Risk estimates for hypothetical use of groundwater as a source of drinking water exceed targets established by EPA. Risk assessment of exposure to groundwater was conducted as part of the future land use analysis for the agricultural, residential, and commercial scenarios. Following EPA guidance in RAGS (EPA 1989e), the assessment was based on the use of unfiltered samples. As a point of comparison, risk assessment also was conducted using filtered samples.

Note that the risk estimates for the groundwater pathway, regardless of creek segment or land use area, are based on a common data set. Groundwater data obtained from all monitoring wells along the length of EFPC were aggregated and used in the risk assessment (i.e., risk estimates were based on the arithmetic mean and UCL estimates of the arithmetic mean).

The results of the groundwater assessment indicate that inorganic contaminants adsorbed to suspended material are largely responsible for the elevated risk estimates. Unfiltered concentrations of mercury, cadmium, and lead (UCLs) exceeded federal MCLs. However, mean and UCL concentrations in filtered samples of groundwater did not exceed MCLs. The observed concentrations of mercury in unfiltered samples resulted in HQ scores exceeding 1. The HQ score for mercury based on filtered samples, however, was 16 times *lower* than that derived for the unfiltered samples and did not exceed 1.

Even though contaminant concentrations were considerably reduced in filtered samples, the overall HI score for the groundwater ingestion pathway still exceeds 1. This is entirely due to dissolved concentrations of manganese. The presence of dissolved arsenic is the reason for the excess lifetime cancer risk estimate for groundwater remaining above 10^{-4}. Manganese is present in background soils at levels comparable to that in EFPC soils, and therefore, naturally occurs in environmental media in this geographic area. Although levels of arsenic in floodplain soils exceed observed background concentrations (Hinds Creek), the presence of arsenic in groundwater cannot clearly be attributed to the Y-12 Plant.

The results of the Monte Carlo analysis of the groundwater pathway indicate a range of variability (i.e., based on uncertainty in exposure assumptions) of less than an order of magnitude. RME risk estimates are very comparable to the 90th percentile estimates, and are two to more than four times greater than the 50th percentile estimates from the probability distributions.

In summary, the groundwater ingestion pathway is concluded to be a minimal concern to residents of EFPC. No one is currently using groundwater in the vicinity of EFPC as a source of drinking water or is likely to do so in the future. The comparison of unfiltered and filtered results demonstrates that most concentrations of inorganic chemicals substantially decreased in the filtered samples. Notable exceptions to this include arsenic and manganese. Note that manganese levels in soil are present at concentrations comparable to background. While arsenic levels exceed background concentrations in soil, there does not appear to be a plausible link relating arsenic to the Y-12 Plant. Exposure point concentrations determined from filtered samples did not exceed MCLs and resulted in considerably reduced risk estimates. Taking into consideration the conservatism of the RME estimate and the uncertainty inherent in the exposure assumptions, the groundwater pathway should *not* be used as a basis for determining the need for site remediation.

5.6.5 Conclusions: Influence of Toxicity of Mercury in the BRA

Monte Carlo simulation has been used to explore the influence of uncertainty in the exposure variables on the estimates of risk to human health. As discussed in Sect. 5.3, considerable uncertainty is associated with the EPA toxicity measures used in the baseline human health risk assessment. Just as the exposure variables are not truly single point values, the toxicity measures should not be characterized as a single, invariant number. The EPA CSFs constitute a 95th percentile upper-bound estimate of the slope of the dose-response curve. The scientific literature is replete with studies exploring the variation in cancer slope estimates that is introduced by the selection and use of high to low dose extrapolation models. The RfDs and RfCs proposed by EPA for use in risk assessment are derived from NOAEL or LOAEL estimates and the application of uncertainty and modifying factors. Depending upon the quality of the experimental study, one to three orders of magnitude "safety" may be built into the RfDs. The uncertainty in these toxicity measures is thus quite high.

Mercury is the predominant COC in the EFPC study area. Section 5.3 and Appendix N provide a comprehensive discussion of the uncertainty surrounding the RfD used in the BRA. The RfD for mercury has been withdrawn from the IRIS data base pending further review. HEAST lists a value of 3×10^{-4} mg/(kg • d) that was used in the EFPC study (EPA 1992e). This value was based on a very sensitive endpoint in laboratory test species and may be inappropriate as a basis for extrapolating toxicity from animal species to humans. In addition, the RfD was derived for soluble mercury species, not the insoluble forms likely to be found in floodplain soils of EFPC.

As part of the EFPC RI, an effort was undertaken jointly by SAIC and ORNL to explore the possibility of deriving alternate RfDs for mercury species found in soils of EFPC. These efforts resulted in estimated RfDs for mercury that are one to two orders of magnitude higher than the interim value proposed by EPA. An understanding was reached with EPA Region IV that no toxicity measures would be used in the BRA unless these measures were fully reviewed and approved by EPA Headquarters and the EPA Environmental Criteria and Assessment Office in Ohio. This agreement has been honored in this report, and the alternate RfD for mercuric sulfide has been submitted to EPA for review. However, the derivation of the alternate RfDs has been very illustrative. It shows that there is another one to two orders of magnitude of conservatism in the RME estimates of risks to human health beyond that characterized in the uncertainty analysis of the exposure variables.

The direct implication of these findings is extremely important. If the most meaningful measure of the oral RfD for mercury at EFPC is on the order of 10^{-3} mg/(kg • d), all noncarcinogenic risk estimates presented in the BRA will correspondingly be reduced by an order of magnitude. These results could obviate the need for remediation in locations dominated by the soil ingestion pathways.

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6. BASELINE ECOLOGICAL RISK ASSESSMENT REPORT

The Federal Facility Agreement (FFA) for the East Fork Poplar Creek (EFPC) site among the U.S. Department of Energy (DOE), the U.S. Environmental Protection Agency (EPA), and the Tennessee Department of Environment and Conservation (TDEC) requires a comprehensive investigation to determine the nature and extent of contamination in the floodplain of EFPC. Under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), there must be a remedial investigation/feasibility study prior to site remediation. This includes performance of a baseline ecological risk assessment (ERA). The ERA evaluates the baseline risk levels to various ecological resources and also determines if and where there is imminent and substantial danger to the animal and plant populations and their habitats. Thus, it represents the risk characterization for current and future conditions of the "no action" alternative required by the National Environmental Policy Act (NEPA). The ERA provides the basis for coordinating with and meeting the requirements of DOE as a Natural Resource Trustee as well as those of cooperating trustees as specified under 43 *CFR* 11, Natural Resource Damage Assessments (NRDAs). This coordination is essential to the development of assessment and measurement endpoints and to the ultimate selection of remediation alternatives in the feasibility study/environmental impact statement (FS/EIS).

The EFPC ERA was conducted in accordance with the ERA plan documents (LWA 1991; Radian 1993a) and follows guidance outlined in *Risk Assessment Guide for Superfund Volume II [RAGS II]: Environmental Evaluation Manual* (EPA 1989a), *Ecological Assessments of Hazardous Waste Sites: A Field and Laboratory Reference Document* (EPA 1989b), and *Framework for Ecological Risk Assessment* (EPA 1992a). Also, the ECO updates, beginning with the 1991 issue (EPA 1991), were used as guidance.

Section 6.1 of the baseline ERA describes the site (the 100-year floodplain) and ecological receptors, identifies ecological hazards, and establishes the goals, breadth, and focus of the ERA. Section 6.2 characterizes the exposure to ecological receptors at the site. An assessment of the effects of contaminants on ecological receptors is made in Sect. 6.3. Section 6.4 characterizes the risks to ecological receptors under existing and projected conditions. A summary of the process and conclusions reached in the ERA is contained in Sect. 6.5.

The ERA process provides an overall picture of the plants, animals, and habitats and provides the decision-maker with a comprehensive view not only of the existing risk but also of the information needed to select those remediation alternatives providing the best ecological

protection. The ecological and human health risk assessments can then be integrated into the baseline risk assessment to develop the remediation alternatives that best meet the remediation goals.

6.1 ECOLOGICAL HAZARD IDENTIFICATION AND PROBLEM FORMULATION

6.1.1 Intent and Scope of Ecological Risk Assessment

The ERA considers the risk to natural resources (or "ecological risk"). The ERA process differs from the human health risk assessment process in that the ERA focuses on non-human animal and plant populations, communities, and habitats rather than on individuals. In the ERA process, individuals are addressed only if they are protected under the Endangered Species Act, are unique, or have such public importance that individual consideration is merited.

The ERA must also address the requirements for DOE as a Natural Resource Trustee as well as those for cooperating trustees as specified under 43 *CFR* 11, NRDAs. Requirements under NEPA must also be addressed, including state and federal agency coordination, public involvement, and evaluation of alternatives. To address these requirements, the EFPC ERA has adopted an ecological approach that addresses contaminant effects on populations and communities within the area of influence of the site. To address CERCLA, NEPA, and NRDA requirements, the ERA evaluates contaminant effects on populations and communities within the area of influence of EFPC. A "weight-of-evidence" assessment of risk is made for indicator species and projected to the overall effect of contaminants on populations and habitats within the ecosystem. The weight-of-evidence approach considers the toxicity of contaminants of concern (COCs), routes of exposure, and observed responses of the indicator species. This provides an overall assessment of risk, which will influence the evaluation of alternatives. Through close coordination with the trustees, this assessment can satisfy the numerous requirements of participants in the ERA process.

As described by Suter and Loar (1992), the weight-of-evidence approach to ecological risk assessment is analogous to a court case in which physical evidence, witness accounts, and expert testimony are considered together to reach a verdict based on the preponderance of evidence. Rather than relying on a single line of evidence, the weight-of-evidence approach ideally incorporates three categories of biological investigation.

First, toxicity testing of water, soil, and sediment to which biota are exposed (e.g., static bioassay using *Ceriodaphnia*) provides direct measurement of effects on animal and plant

"indicator" species (see below). Second, biological surveys assess the current state of biota on the site (i.e., field-observed effects) and the results can be compared to a reference site or to the observations expected in the absence of contamination. Finally, body burdens of site-related contaminants can be measured as evidence of exposure and possible effects.

Each single line of evidence has its limitations. For example, toxicity testing is performed under controlled conditions but has shortcomings in that it does not mimic all of the antagonistic/synergistic processes taking place in natural systems. Conversely, biological surveys and body burden measurements are realistic observations of true conditions at the site, but the lack of control afforded by laboratory conditions may weaken the link between cause and effect, because observed conditions could be caused by something other than contaminants at the site. Regardless, the preponderance of evidence from the combined studies can be used to support a statement about the ecological risks posed by site-related contaminants.

All three categories of investigation and their resulting lines of evidence were used in the EFPC ERA. For example, results of standard media toxicity studies done by the Y-12 Plant Biological Monitoring and Abatement Program (BMAP) are described in Sect. 6.3.2.1. Experimental studies of soil toxicity to mice are summarized in Sect. 6.3.2.2, and studies of water and sediment toxicity to aquatic biota are reported in Sect. 6.3.2.3. Biological survey data are described in Sect. 6.3.3, and body burden analyses are reported in Sect. 6.2.3. These lines of evidence are examined with the weight-of-evidence approach, which is explained in greater detail, in Sect. 6.4. for both aquatic and terrestrial indicator organisms.

Another key activity in the ERA is the use of indicator organisms. There are insufficient time and money to study the multitude of species living in the EFPC floodplain. A process is followed to select representatives from each of the major aquatic and terrestrial habitats. These representatives or "indicators" have sufficient similarities to other species (not measured) in the habitat that generalizations can be made about effects to and protection of the indicator that can be used simultaneously to assess and protect the unmeasured species. This approach is explained in Sect. 6.1.4.3 where the justifications are given for the following types of indicator organisms: fish, benthic macroinvertebrates, periphyton, mammals, birds, insects, worms, and vegetation. This indicator organism approach is done in the context of habitats or units of nature where the indicator populations live.

6.1.2 Objectives of Ecological Risk Assessment

The baseline ERA will characterize baseline risk levels throughout the EFPC floodplain and evaluate if and where there is conclusive evidence of imminent and substantial danger or chronic risks to animal and plant populations and habitats due to activities at the Y-12 Plant and from other activities. This information will be used in the feasibility study to decide if and where site remediation may be required.

According to the *Framework for Ecological Risk Assessment* (EPA 1992a), the ERA process (Fig. 6.1) consists of three interrelated phases: problem formulation, analysis, (comprised of exposure characterization and ecological effects characterization), and risk characterization. In performing the ERA, these three phases are realized as the four steps defined below.

Problem formulation provides a preliminary characterization of chemical and physical stressors present in the ecosystem, the components of the ecosystem likely to be at risk, and the potential ecological effects, along with a selection of assessment and measurement endpoints as a basis for a conceptual model of stressors, components, and effects (Sect. 6.1).

Exposure characterization describes the biotic and abiotic ecosystem attributes, along with the route, magnitude, frequency, duration, trend, and spatial pattern of exposure of each indicator population or habitat component in relation to a chemical or physical stressor (Sect. 6.2).

Effects characterization evaluates the ecological response to chemical and physical stressors in terms of the selected assessment and measurement endpoints and, depending on the parameters of exposure, results in a profile of response to stressors at concentrations or doses or other units of stress to which populations and habitats are exposed (Sect. 6.3).

Risk characterization integrates the effects of exposure and stressor response on indicator populations or habitat components, summarizes risk according to the weight of evidence, and then interprets the ecological significance, including the potential for ecosystem recovery (Sect. 6.4).

Specific objectives of the sampling and analysis of indicator populations and their associated habitats and environmental media were:

- to quantify the population levels and community composition at various locations along EFPC and at non-impacted reference sites;
- to measure the concentrations of contaminants of ecological concern (COCs) in EFPC surface water, sediments, and soils;

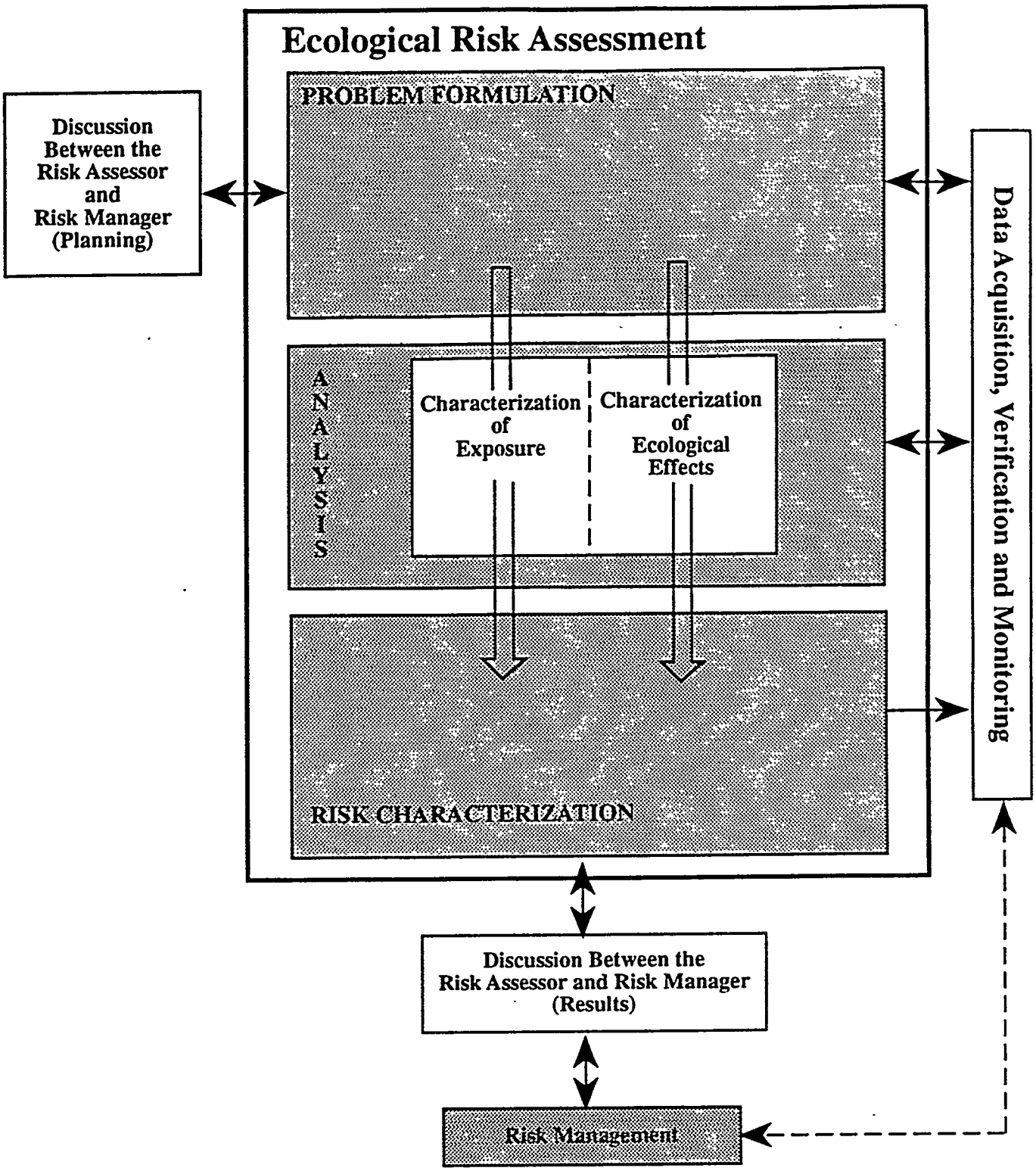


Fig. 6.1. Framework for Ecological Risk Assessment. *Source:* EPA (1992).

- to measure the concentrations of ecological concern (COCs) in aquatic (instream) and terrestrial (floodplain) indicator biota and the environmental media;

- to determine the presence in the EFPC floodplain of species designated as threatened or endangered, and of protected or unique habitats (i.e., wetlands);

- to develop relevant scenarios for realistic exposure of organisms at higher trophic levels to contaminants via the food web;

- to display these findings using maps of contaminant distribution, habitats of indicator populations, and stressor response (mortality, pathology, reproduction, or growth); and

- to integrate these findings using a weight-of-evidence approach.

The integrated findings of the ERA provide information to help select remediation alternatives in the FS/EIS process.

6.1.3 Ecological Assessment and Measurement Endpoints

Ecological assessment endpoints are goals or objectives for environmental values to be protected during the remediation process. For example, the U.S. Fish and Wildlife Service (Eisler), which serves as the trustee for migratory birds, may establish objectives for their protection. Indicator species representing these birds and their food resources and physical environment variables can be selected (1) for investigation to determine if migratory birds are or may potentially be affected negatively by contamination at a site and (2) to support the evaluation of alternatives. Where assessment endpoints are not directly measurable, one or more measurement endpoints were chosen to determine, either qualitatively or quantitatively, site-specific impacts on the assessment endpoints. Assessment policy goals and measurement endpoints described below were developed within the context of guidance in EPA (1992a) and Suter (1993).

The following assessment endpoints are relevant to the remediation of EFPC. Measurement endpoints are indicated as are any legislation, executive orders, regulations, or guidelines that were directly relevant to the endpoint. Some policy goals and assessment endpoints have less exact legislative authority and are technical interpretations of the broad requirement to protect and restore the environment associated with CERCLA and NEPA. Assessment and measurement endpoints are provided within the context of policy goals.

Policy Goal 1: The conservation of threatened, endangered, and rare species and their critical habitats.

Assessment Endpoint 1a: No harm to any state- or federally-designated threatened or endangered species and their critical habitats on the EFPC floodplain.

Measurement Endpoint 1a: Surveys of the presence or absence of species and their habitats on the EFPC floodplain.

Assessment Endpoint 1b: Maintenance of plant community composition and/or structure required for rare plant and animal and other support species.

Measurement Endpoint 1b: The abundance and distribution of plant and animal species that would support threatened, endangered, and rare species.

Legislation: The Endangered Species Act of 1973, the Tennessee Non-Game and Endangered or Threatened Wildlife Species Conservation Act of 1974, and the Tennessee Rare Plant Protection and Conservation Act are relevant. Also, CERCLA and NEPA provide authority and a process to protect these ecological resources.

Policy Goal 2: The protection of migratory birds.

Assessment Endpoint: No killing or harming of migratory birds as a result of exposure to site-specific stressors.

Measurement Endpoint: Contaminant concentrations in selected EFPC food sources for migratory birds and weight-of-evidence data.

Legislation: The Migratory Bird Treaty Act (16 *USC* 703–711). Also, CERCLA and NEPA provide authority and a process to protect these ecological resources.

Policy Goal 3: The preservation of wetlands.

Assessment Endpoint: The presence and structure/function of wetlands in relation to contaminants.

Measurement Endpoint: Surveys of wetlands and associated contamination levels, if any, on the EFPC floodplain.

Related Executive Orders and Implementing Regulations: Relevant standards are Executive Orders 11988 and 11990 of 1979, and 10 *CFR* 1022 establishing DOE regulations of compliance with floodplains/wetlands environmental review requirements

[Federal Register 44 (46): 12594-12599]. Also, CERCLA and NEPA provide authority and a process to protect these ecological resources.

Policy Goal 4: The existence of a fish community indicative of undegraded conditions.

Assessment Endpoint: A fish community in which the proportion of species classified as tolerant of degraded water quality conditions (Ohio EPA 1989) is less than 30% (approximately the proportion of tolerant species at the reference site).

Measurement Endpoint: The measurement endpoint is the proportion of tolerant species to the overall populations at the site.

Legislation: Relevant authorities are CERCLA and NEPA for protection of the environment.

Policy Goal 5: No adverse effects from contaminants to aquatic indicator organisms and/or predators that feed on them.

Assessment Endpoint 5a: The ratio of concentrations of contaminants in surface water to water quality criteria for protection of aquatic life should be ≤ 1.

Measurement Endpoint 5a: Contaminants in surface water.

Assessment Endpoint 5b: The ratio of body burdens of contaminants in indicator organisms to levels expected to cause toxicological effects should be ≤ 1.

Measurement Endpoint 5b: COC concentrations in whole body samples of indicator species.

Assessment Endpoint 5c: The ratio of body burdens of contaminants in fish to levels expected to be protective of piscivorous biota should be ≤ 1.

Measurement Endpoint 5c: COC concentrations in whole-body samples of aquatic indicator prey species.

Legislation: Relevant authorities are CERCLA and NEPA for protection of the environment.

Policy Goal 6: The existence of a terrestrial animal community indicative of undegraded conditions.

Assessment Endpoint 6a: Diversity, abundances, and distributions of terrestrial animals should not result in a $\geq 20\%$ decrease compared to the reference.

Measurement Endpoint 6b: The measurement endpoint is population abundance and distribution by habitat.

Legislation: Relevant authorities are CERCLA and NEPA for protection of the environment.

Policy Goal 7: No adverse effects from contaminants to terrestrial indicator organisms and/or predators that feed on them.

Assessment Endpoint 7a: The ratio of body burdens of contaminants in indicator organisms to levels expected to cause toxicological effects should be ≤ 1.

Measurement Endpoint 7a: COC concentration in whole-body samples of terrestrial indicator species.

Assessment Endpoint 7b: The ratio of body burdens of contaminants in indicator organisms to levels expected to be protective of terrestrial predators should be ≤ 1.

Measurement Endpoint 7b: COC concentration in whole-body samples of terrestrial indicator prey species.

Legislation: Relevant authorities are CERCLA and NEPA for protection of the environment.

6.1.4 Environmental Description

This subsection describes the sampling sites used for ecological field investigations and ecotoxicological studies of the lower EFPC study area. The field investigations were conducted by field teams from Science Applications International Corporation and Radian Corporation during September through December 1991.

6.1.4.1 Spatial extent of study area

The lower EFPC study area originates at the discharge point of Lake Reality on the Y-12 Plant site and continues for ~24 km (15 miles) before reaching EFPC's confluence with Poplar Creek. The creek travels through (a) engineered channels starting below the National Oceanic and Atmospheric Administration (NOAA) Site, (b) commercial and residential areas in the city

of Oak Ridge, (c) privately-owned forest and pasture lands for the majority of its length, and (d) the Oak Ridge Reservation (ORR) for the last 7.4 km (4.6 miles) prior to its confluence with Poplar Creek. The Sewer Line Beltway (SLB)—a buried pipeline in Oak Ridge—for which backfill was obtained from the EFPC floodplain is discussed in the Exposure Assessment (Sect. 3.3.10).

EFPC sampling site locations

Ecological sampling sites were chosen to gain maximum information on EFPC biota and physical environment and to allow comparison and contrast with historical data from studies conducted by Van Winkle et al. (1984), the Tennessee Valley Authority (TVA) (1985), and the Y-12 BMAP (Loar 1992; Hinzman 1992). These studies are described in Sect. 6.2.3.1. Figure 6.2 shows the location of the various sampling sites and Table 6.1 presents the sampling site locations by kilometer along EFPC for these studies.

Six primary sites were selected on EFPC, five of which are relatively close to the sampling sites for the Y-12 Plant BMAP (Loar 1992; Hinzman 1992). Communication and coordination with BMAP sampling personnel ensured that no adverse effects occurred to those sampling locations or to the BMAP long-term studies. Locations were chosen that were believed to be physically and biologically similar to one another. For example, the stream sampling locations had a similar mixture of riffle, run, and pool areas and sunlight exposure. This facilitates comparisons to the BMAP work. Locations were chosen that were believed to be physically and biologically similar to one another. For example, the stream sampling locations had a similar mixture of riffle, run, and pool areas and sunlight exposure.

Sites were selected based on their relationship to known sources of contamination. Two sites were located in the upper reaches of EFPC, one in Pine Ridge Gap near the outfall from Lake Reality and near the BMAP sampling location at East Fork kilometer (EFK) 23.4, and one at the contaminated EFPC Remedial Investigation (RI) Phase Ia Site NO behind the NOAA facility. At the latter site, groundwater, surface water, instream sediments, and soil cores have been sampled; the analytical data from these samples provide a background for the interpretation of findings from ecological surveys and tests. Similarly, a location near the contaminated Phase Ia Site BR (EFK 17.6) was chosen to take advantage of previous sampling and its proximity to the BMAP sampling location at EFK 18.2. This site serves as an upstream reference for impacts of the publicly owned treatment works. A location was chosen below the publicly owned treatment works outfall to assess impacts of the treated municipal wastewater on

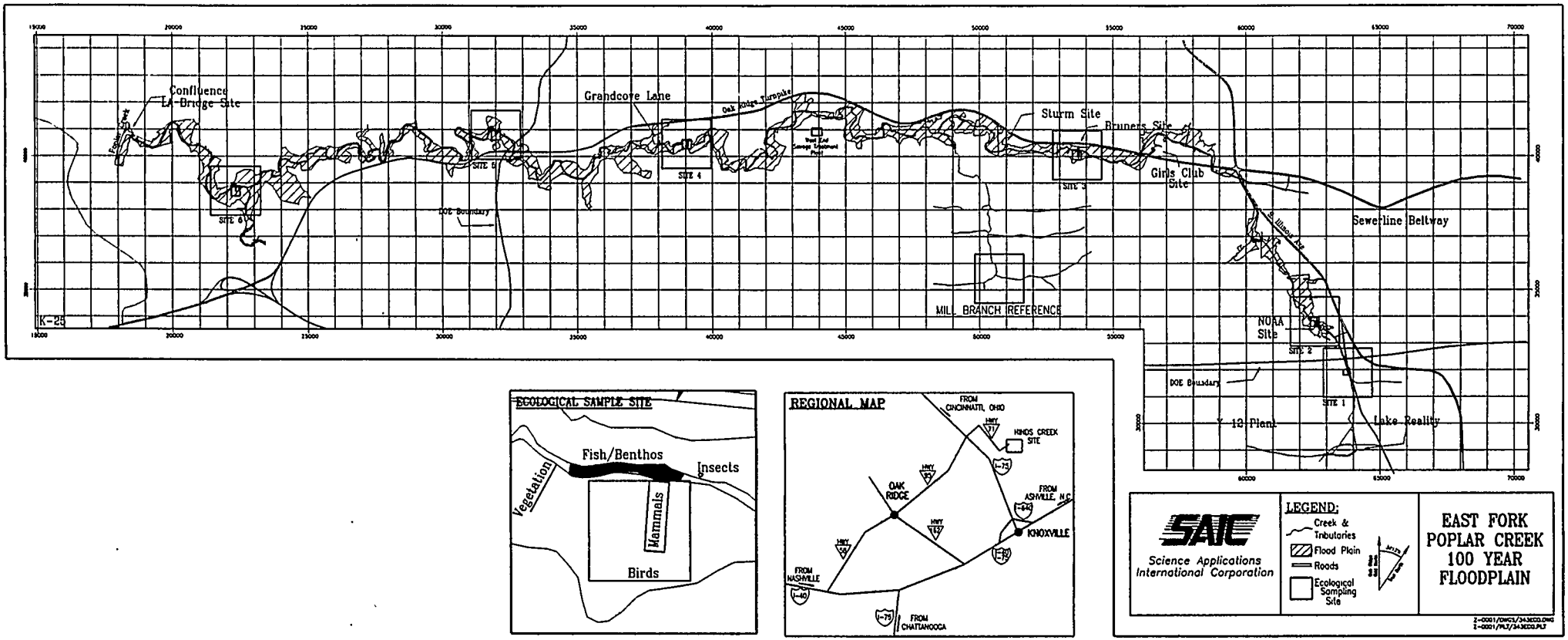


Fig. 6.2. Map of EFPC sampling sites.

Table 6.1. Historical sampling locations for ecological measurements on lower EFPC, 1982 to 1991

EFK	Measuring entity			
	Van Winkle et al.[a]	TVA[b]	BMAP[c,d]	EFPC ERA
23.4			MT,B,P	
23.2			B	
23.1			MT	
22.8	B		MT,A	(Site 1) B,P
22.7	B			
22.2	B	B		
22.0				(Site 2) B,P
21.9			MT,A	
20.5			MT,A	
18.2			MT,B,A,P	
17.6				(Site 3) B,P
17.0			B	
16.1			MT,A	
14.2		B		
14.0			MT	
13.8			MT,B,A,P	
13.4	B			
10.9			MT,A	
10.8				(Site 4) B,P
10.6			B,P	
10.0			P	
8.8	B			
7.7	B			
7.6			MT,A	

Table 6.1 (continued)

EFK	Measuring entity			
	Van Winkle et al.[a]	TVA[b]	BMAP[c,d]	EFPC ERA
7.3				(Site 5) B,P
6.4		B		
6.3			B,P	
5.1			MT,A	
2.3				(Site 6) B,P
2.1	B		MT,B	
Total number of sites	7	3	18	6

Note: A = Algae Photosynthesis Study
B = Bioaccumulation Studies
MT = Media Toxicity Tests
P = Population Surveys

[a]Van Winkle et al. 1984
[b]TVA 1985
[c]Loar 1992
[d]Hinzman 1992

the biota. Two locations were chosen in the lower reaches on the ORR, one above and one below the confluence with Bear Creek.

A final selection criterion was access. Sites were selected to allow easy as opposed to difficult access for sampling gear and samplers. Thus, the following six sites were selected according to various criteria.

Site 1. Site 1 (Fig. 6.3) is located in Pine Ridge Gap at EFK 22.8 downstream of the National Pollutant Discharge Elimination System (NPDES) outfall from Lake Reality near the BMAP sampling location at EFK 23.4. At this site, EFPC is a stream, contained in a well-defined channel, and is about 6 m (20 ft) wide, having minimal floodplain development. At Pine Ridge Gap, the presence of rock outcrops along the west bank indicates a potential link to the groundwater system (Welch 1989). The creek bottom is graded from a rocky limestone rip-rap below Lake Reality to a somewhat smooth and muddy floor beginning just beyond Pine Ridge. The forest type for this site is mixed mesophytic dominated by boxelder (*Acer negundo*) and redbud (*Cercis canadensis*). The primary soil types found in this area are Newark silt loam, Lehew loam, and Armuchee-Muskingum complex (Moneymaker 1981).

Site 2. Site 2 (Fig. 6.4) is located at EFK 22.0 in the vicinity of the contaminated Phase Ia Site NO behind the NOAA facility. At this site, EFPC consists of several braided stream sections of varying widths. The braided sections join into one channel at the lower end of the sample area where the fish sampling survey was conducted. In this reach, the channel has a muddy bottom, and average width is ~4.6 to 6.1 m (15 to 20 ft). The floodplain is ~150 m (492 ft) wide and contains deep-rooted macrophytes. The forest type for this site is mixed mesophytic dominated by green ash (*Fraxinus pennsylvanica*) and ironwood (*Carpinus caroliniana*). Floodplain soils in this area are Newark silt loam (Moneymaker 1981).

Site 3. Site 3 (Fig. 6.5) is located at EFK 17.6 near the Phase Ia Site BR. It is close to the BMAP sampling location at EFK 18.2 and serves as an upstream reference site for any impacts of the Oak Ridge publicly owned treatment works. The EFPC floodplain has been disturbed in this reach as a result of construction activities associated with the SLB and Oak Ridge Turnpike. The bottom of the stream channel consists of mud and rocks and/or gravel. The channel is about 4.6 to 6.1 m (15 to 20 ft) wide. The floodplain, ~130 m (427 ft) wide, contains large sediment deposits. Deep-rooted vegetation is common and rock outcrop is present on the south side of Oak Ridge Turnpike. Site 3 comprises two distinct terrestrial habitats—a bottomland hardwood forest and an old field. Boxelder provides the greatest percent cover,

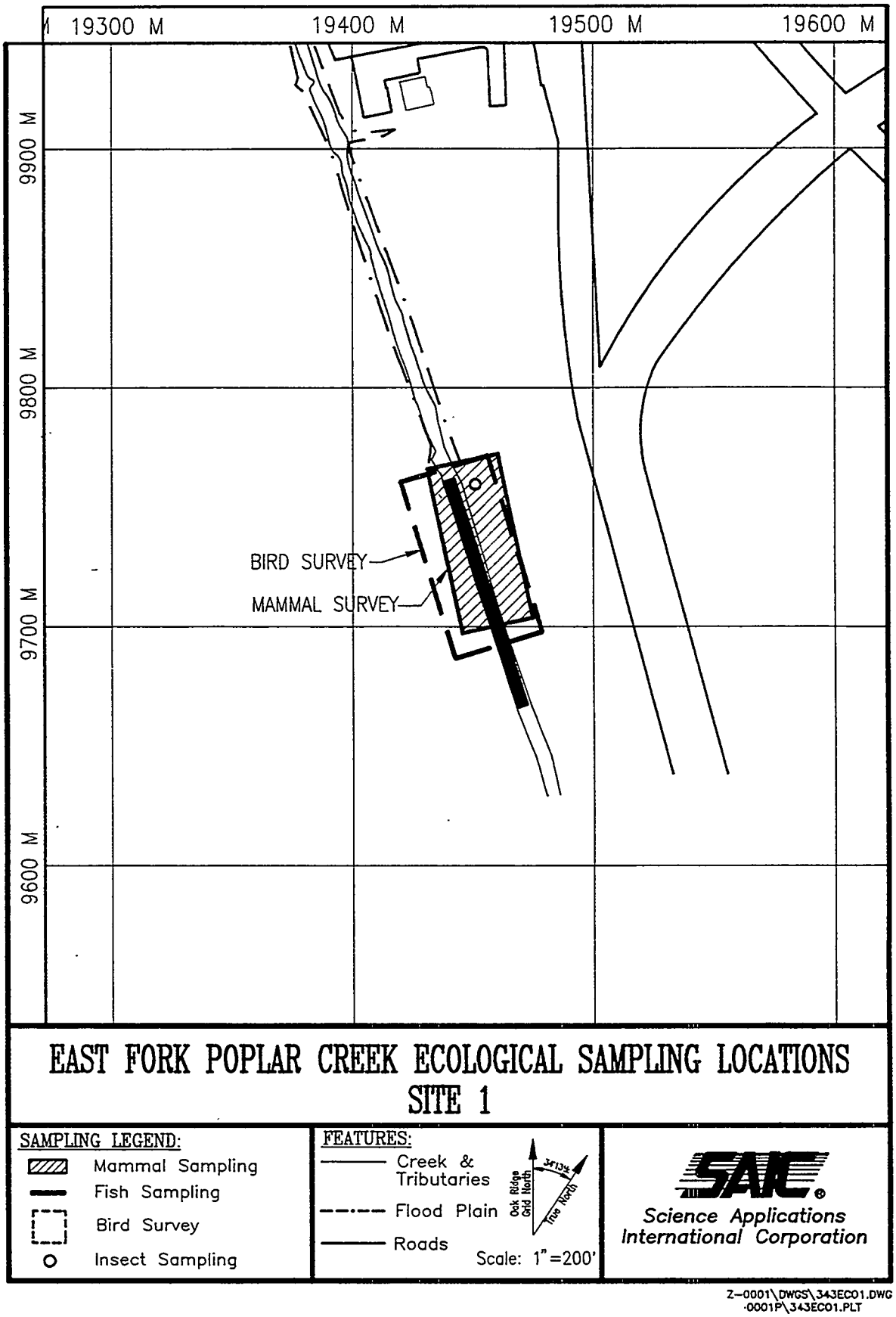


Fig. 6.3. EFPC ecological sampling locations, Site 1.

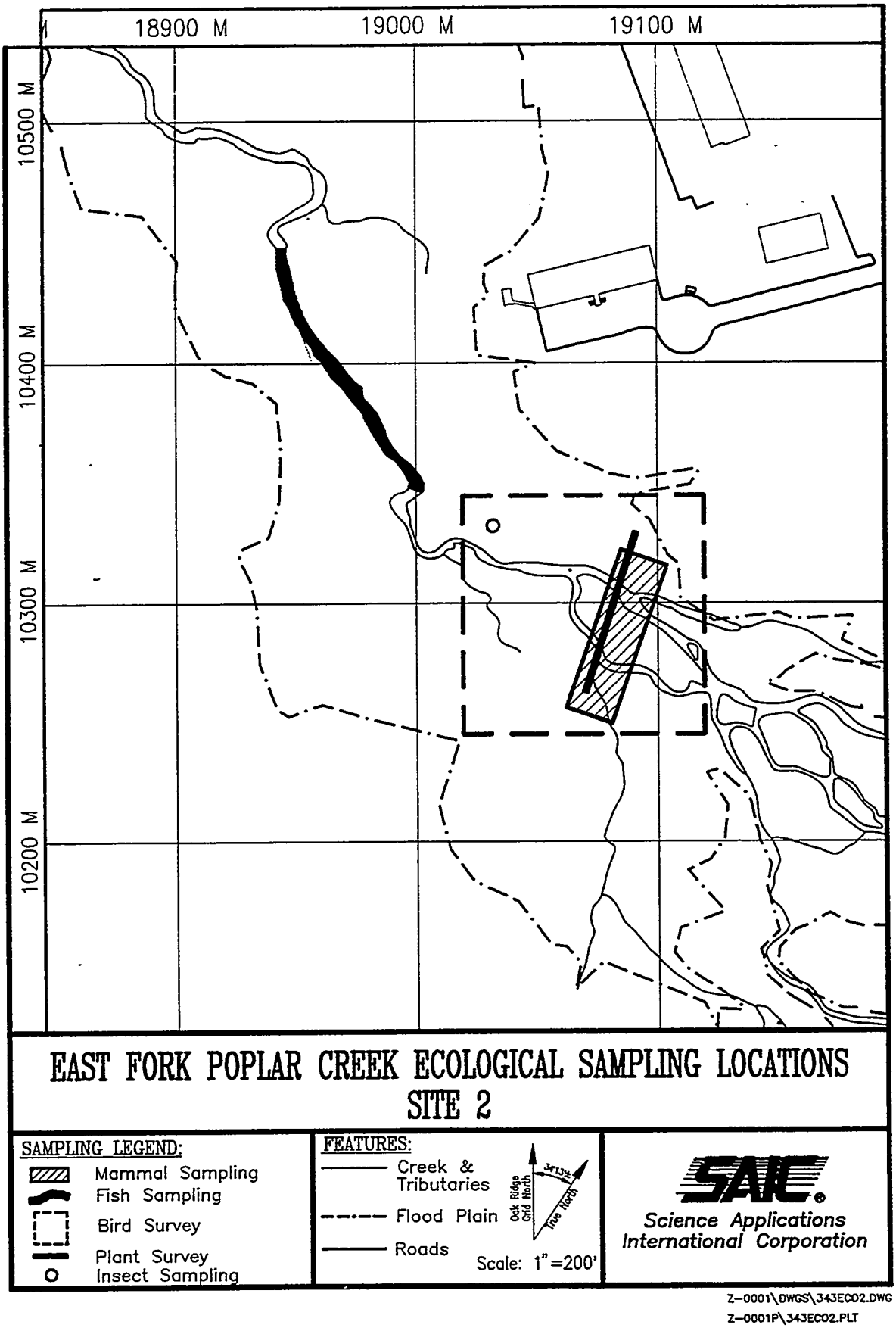


Fig. 6.4. EFPC ecological sampling locations, Site 2.

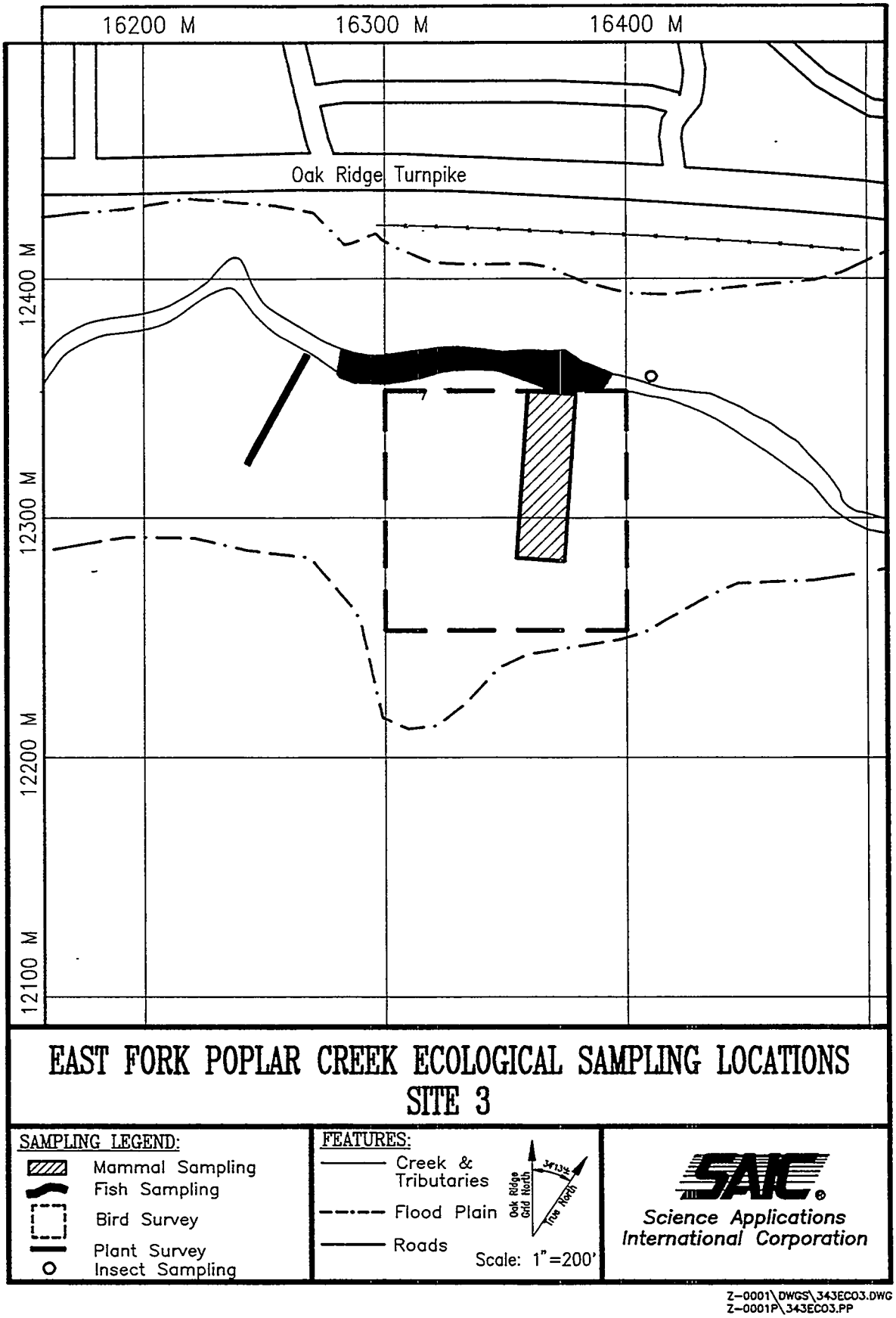


Fig. 6.5. EFPC ecological sampling locations, Site 3.

relative cover, and relative abundance at the site. Floodplain soils are Newark silt loam (Moneymaker 1981).

Site 4. Site 4 (Fig. 6.6) is located at ~EFK 10.8 just downstream from the bridge on Gum Hollow Road near the BMAP sampling location at EFK 10.6. A residential area is located on the north side of the creek, and the Oak Ridge Golf and Country Club is located to the south. This site was chosen because it is ~1.6 km (1 mile) below the publicly owned treatment works outfall and can be used to assess the joint impacts of treated municipal wastewater and Y-12 Plant contamination on biota. At this site the creek is about 3 to 4.6 m (10 to 15 ft) wide. Deep-rooted vegetation is present on the site, and the creek bottom consists of mud and gravel. In this area, rock outcrop can be observed along the south bank. The forest habitat type for this site is bottomland hardwood; boxelder provides the greatest percent cover, relative cover, and relative abundance. The floodplain is about 100 m (328 ft) wide and is generally composed of Roane gravelly loam (Swann et al. 1942)..

Site 5. Site 5 (Fig. 6.7) is located on the lower reach of EFPC near EFK 7.3. The site is located upstream from the confluence of EFPC with Bear Creek. This section of the creek flows through predominantly undeveloped woodland. The creek is about 6.1 to 9.2 m (20 to 30 ft) wide and has a muddy to gravelly bottom. The width of the floodplain at this site is ~200 m (656 ft) wide. Boxelder provides the greatest percent cover, relative cover, and relative abundance in the bottomland forest surrounding the site. Floodplain soils in this area are mapped as Roane gravelly loam (Swann et al. 1942).

Site 6. Site 6 (Fig. 6.8) is located at EFK 2.3 in the lower reach of EFPC, just below the confluence with Bear Creek. The creek in this reach is about 15.2 to 18.3 m (50 to 60 ft) wide and has a muddy to gravelly bottom. The floodplain is heavily vegetated at elevations above the normal water level and extends more than 200 m (656 ft) on the northwest side of the creek and even farther on the south. Pooling caused by high water levels in Watts Bar Reservoir is often evident in this reach. Bottomland hardwood forests surround this site. Boxelder provides the greatest percent cover, relative cover, and relative abundance. Floodplain soils are mapped as Pope very fine sandy loam (Swann et al. 1942).

Reference site locations

The EFPC watershed is not typical of most impaired lotic ecosystems because there is no unaffected upstream location that can be used as an unimpacted reference or control site. Therefore, an alternative was sought. After a careful consideration of all the information available on surface and subsurface geology, hydrology, anthropogenic sources, and residue

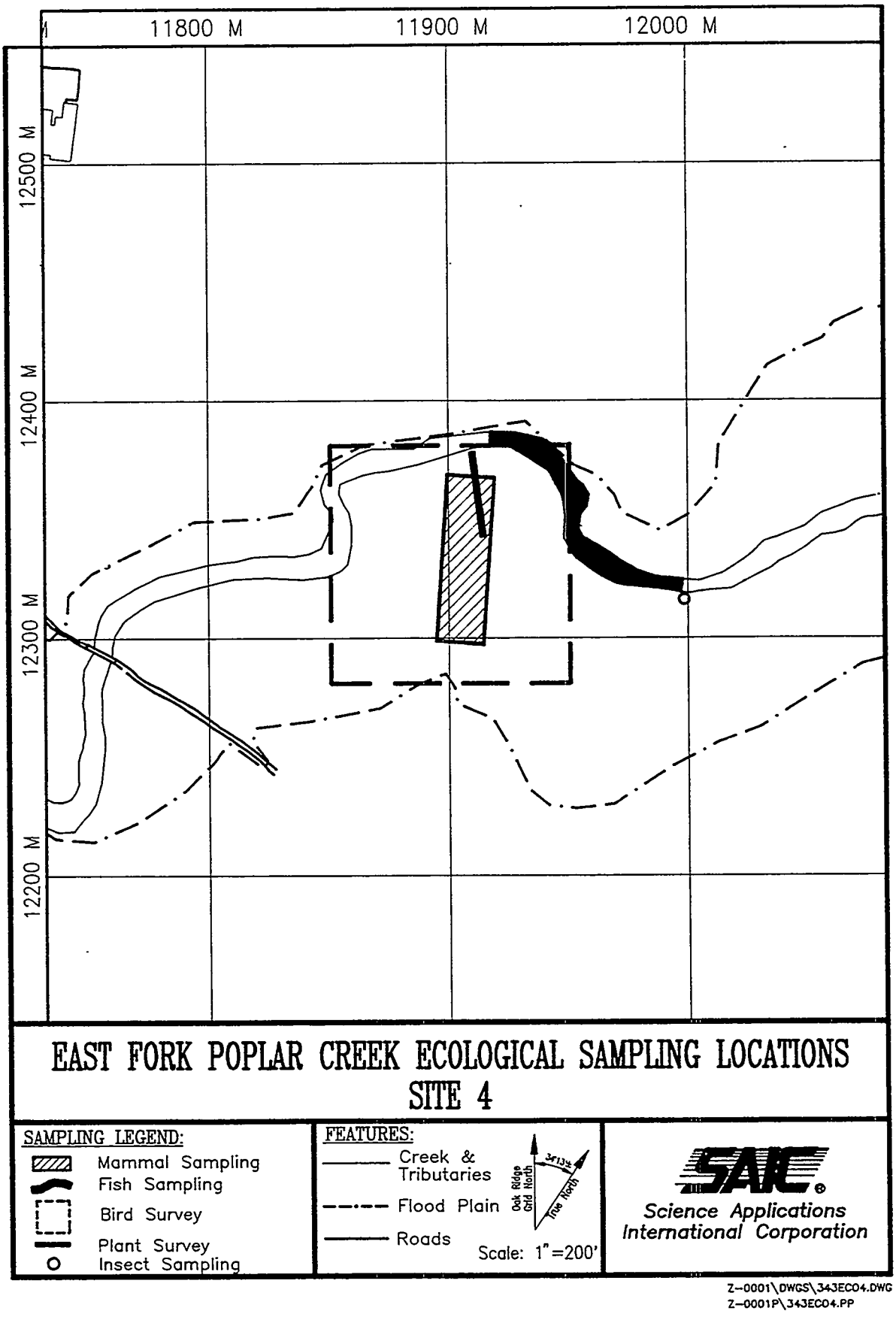


Fig. 6.6. EFPC ecological sampling locations, Site 4.

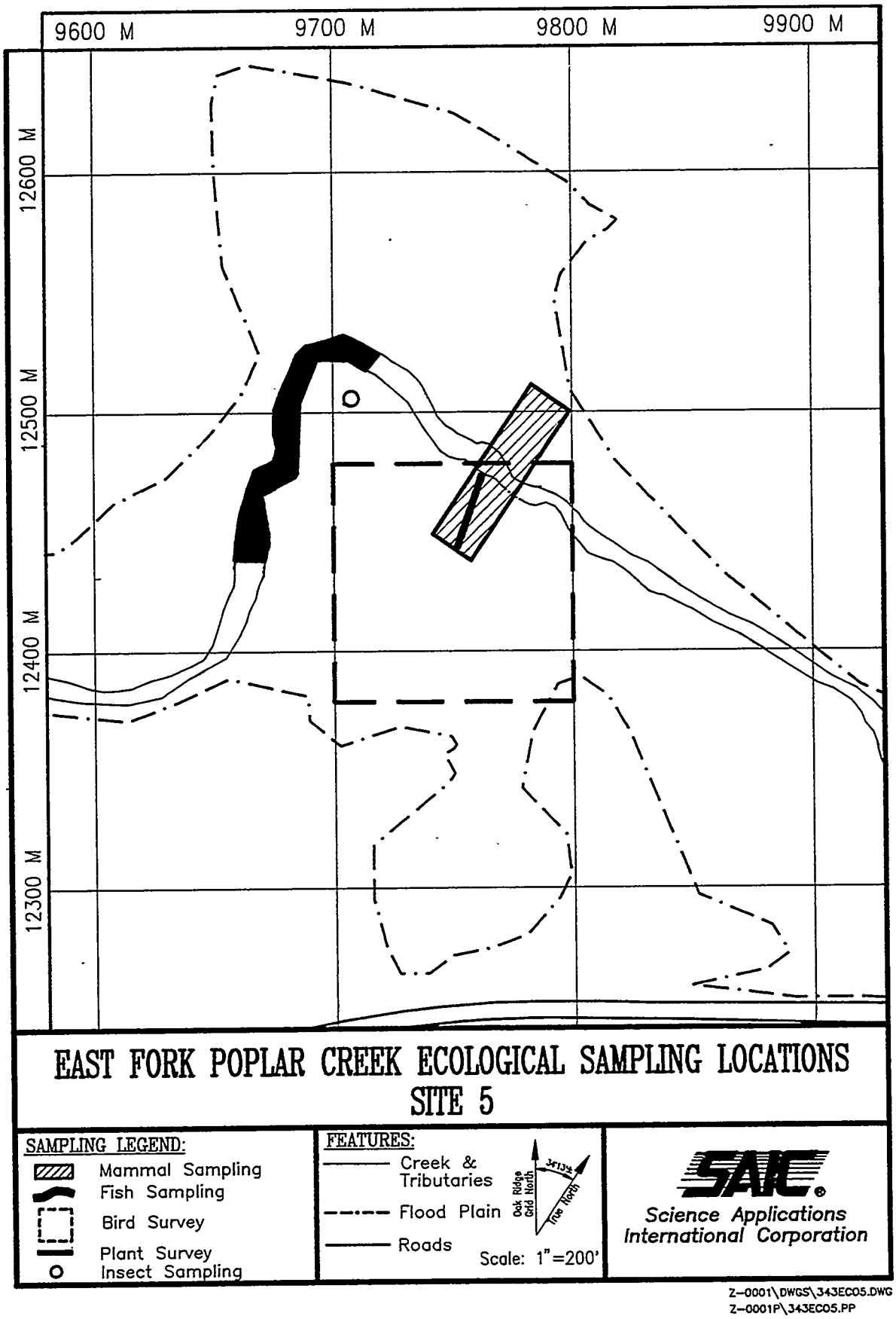


Fig. 6.7. EFPC ecological sampling locations, Site 5.

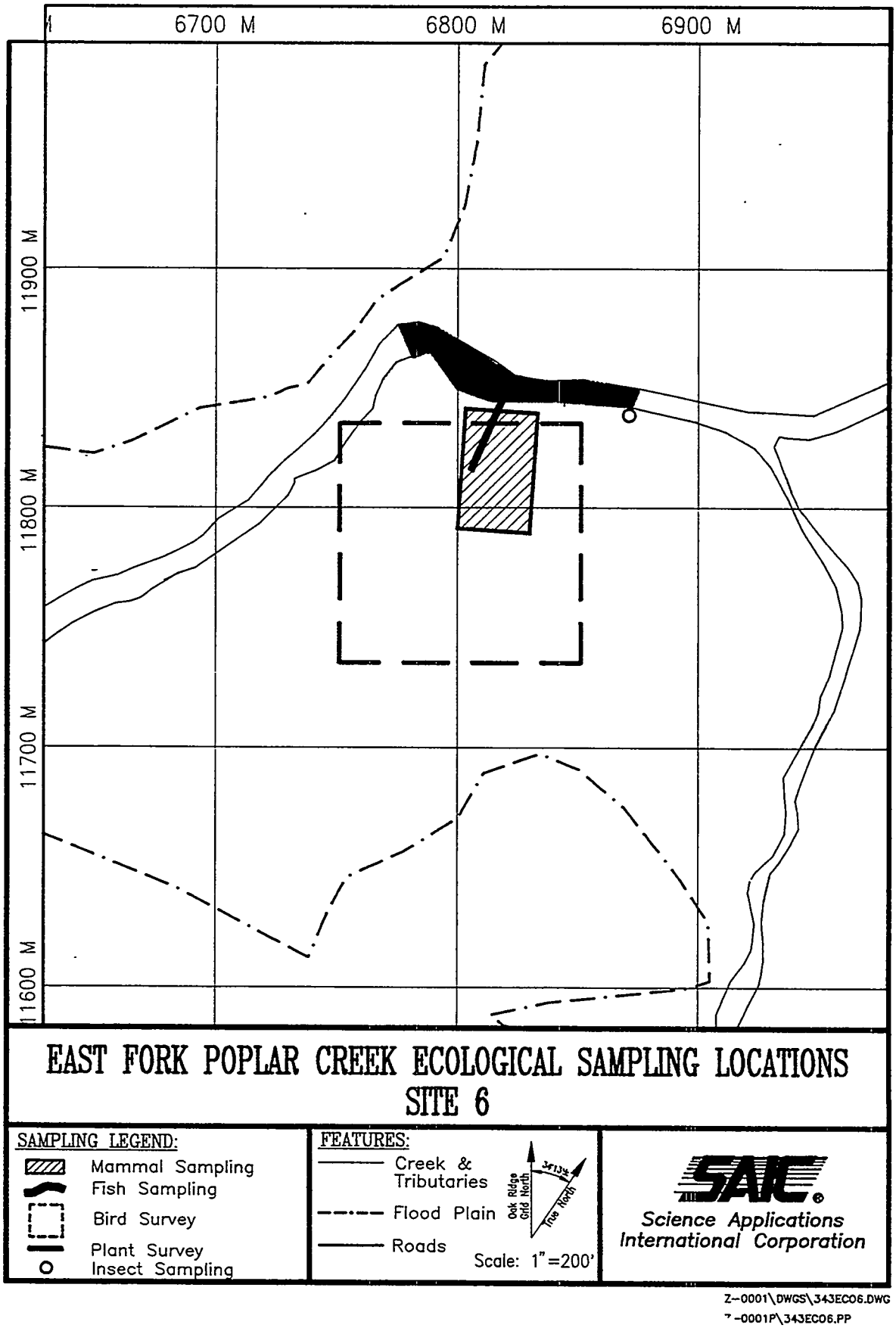


Fig. 6.8. EFPC ecological sampling locations, Site 6.

analyses in fish, Hinds Creek (a relatively unaffected watershed in the Clinch River system) was chosen as the reference site most closely approximating the EFPC watershed.

Although the Hinds Creek reference site was adequate for sampling aquatic biota, it was not a good reference site for terrestrial biota. Topography and land use around the site were atypical of patterns found along most of EFPC. Because of this, an alternative reference site was chosen on Mill Branch, a major tributary in the EFPC watershed. However, the Mill Branch reference site was also not a completely suitable reference area in terms of species compositions for the terrestrial biotic study. The Mill Branch reference site has a more upland community vegetation with an immature forest unlike the bottomland hardwood sites on EFPC. As a result, comparisons with areas similar to EFPC in terms of species compositions were hindered by the lack of a completely suitable reference area for the terrestrial biota aspect of the study. However, for chemical reference purposes – a major part of this ERA – the Mill Branch reference location is suitable.

Hinds Creek Reference Site. The Hinds Creek site (Fig. 6.9) is located ~5.8 km (3.6 miles) from Highway 441, south of Norris, Tennessee, along Hinds Creek Road. Mean width of the creek along the sampling reach was ~5.7 m (18.7 ft), and it has a gravelly bottom. On the north side of the creek, pasture lies between the creek and the road, and the opposite bank forms the base of Chestnut Ridge. Soils at this site along Hinds Creek are Hamblen silt loam (Moneymaker 1981). This reference site was believed to be relatively chemically uncontaminated and, therefore, totally adequate for making comparisons to chemically contaminated sites. In addition, resident fauna were thought to be relatively impaired.

Mill Branch Reference Site. The Mill Branch site (Fig. 6.10) is located ~2.2 km (1.4 miles) upstream from the confluence of Mill Branch with EFPC. The forest at this site is mixed mesophytic and is dominated by sweetgum (*Liquidambar styraciflua*), Virginia pine (*Pinus virginiana*), and dogwood (*Cornus florida*). These species and habitats were similar to those at EFPC. There were no old-fields at this site. Soils at this site along Mill Branch are Hamblen silt loam (Moneymaker 1981). This reference site was believed to be relatively chemically uncontaminated and, therefore, totally adequate for making comparisons to chemically contaminated sites.

Browse and Garden Study

Grasses and vegetables were sampled for both human health risk and ecological risk assessments. Grasses were sampled at Site 3 (Fig. 6.11), Site 7 (Fig. 6.12), and Site 8 (Fig. 6.13) (mercury concentrations shown on these figures are those at the nearest soil sampling

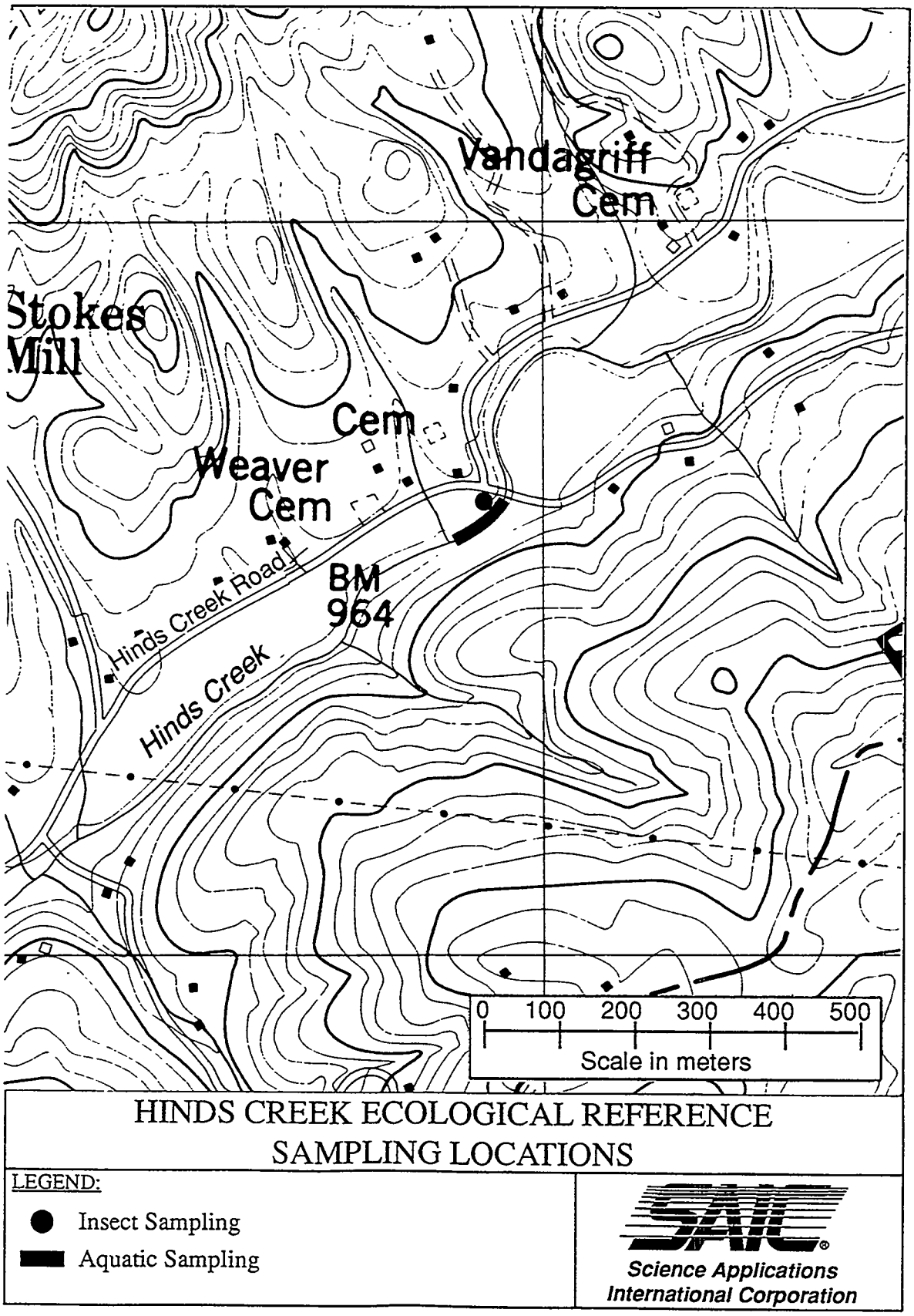


Fig. 6.9. Hinds Creek ecological reference sampling sites.

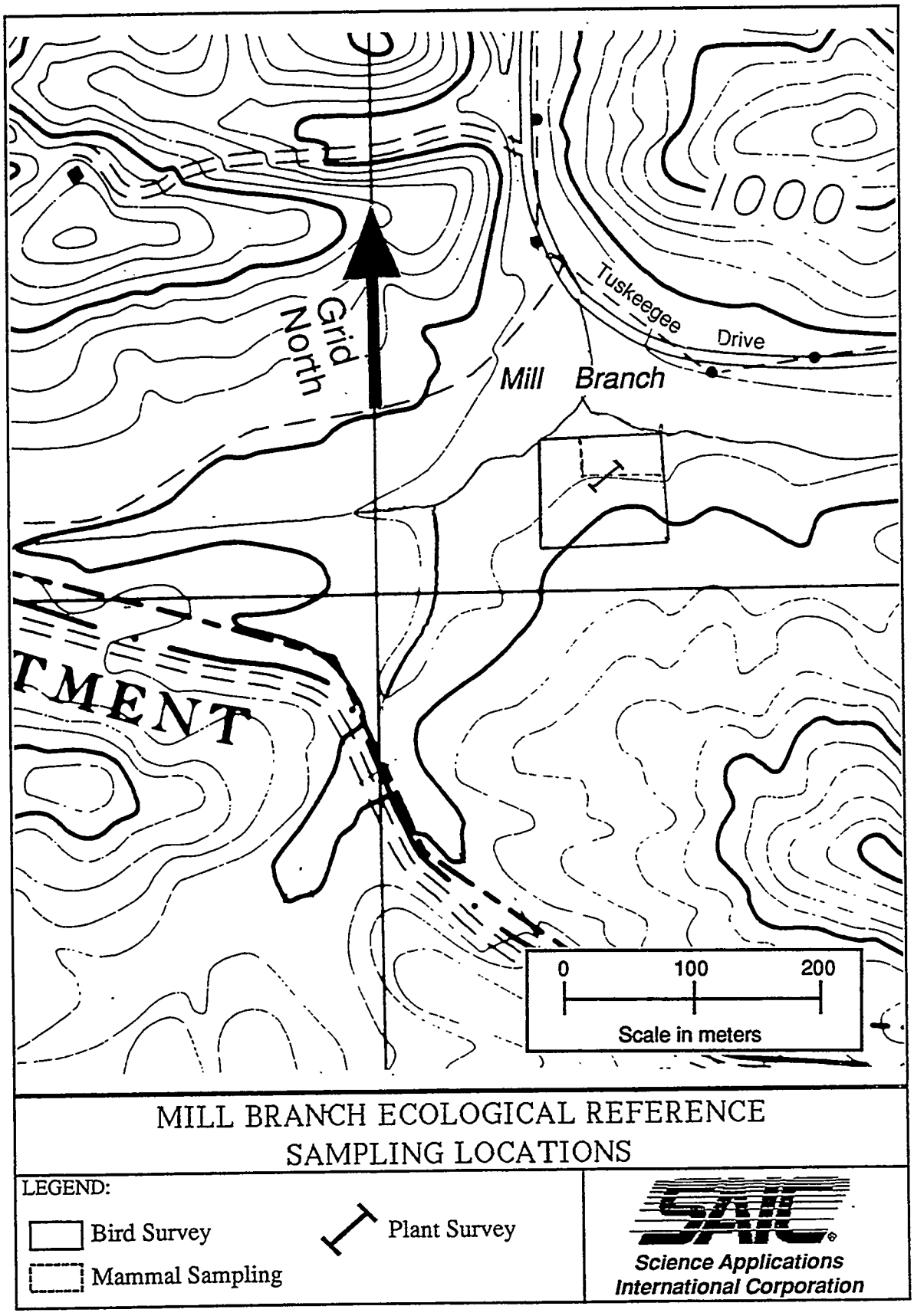


Fig. 6.10. Mill Branch ecological reference sampling sites.

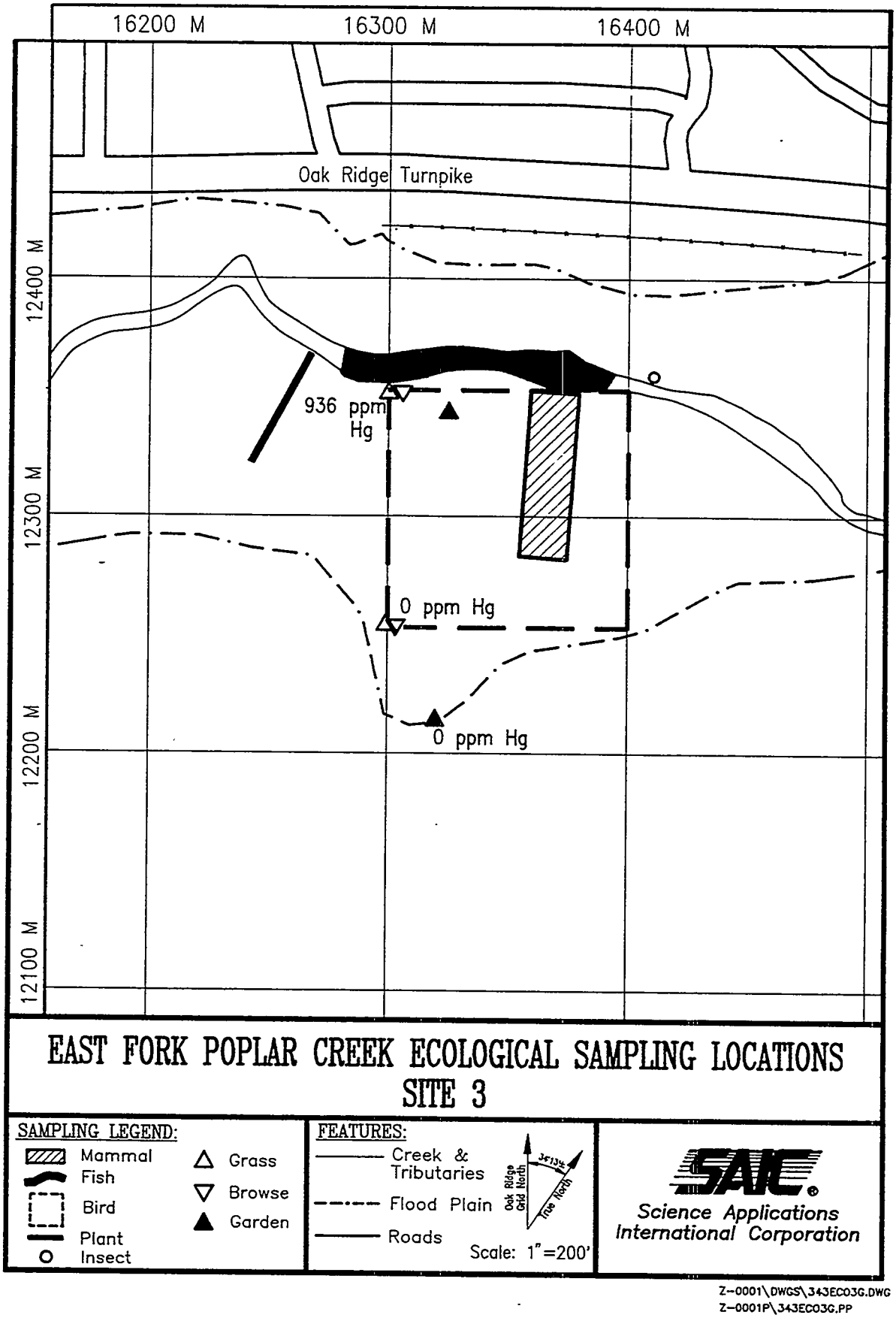


Fig. 6.11. EFPC sampling locations for grasses at Site 3.

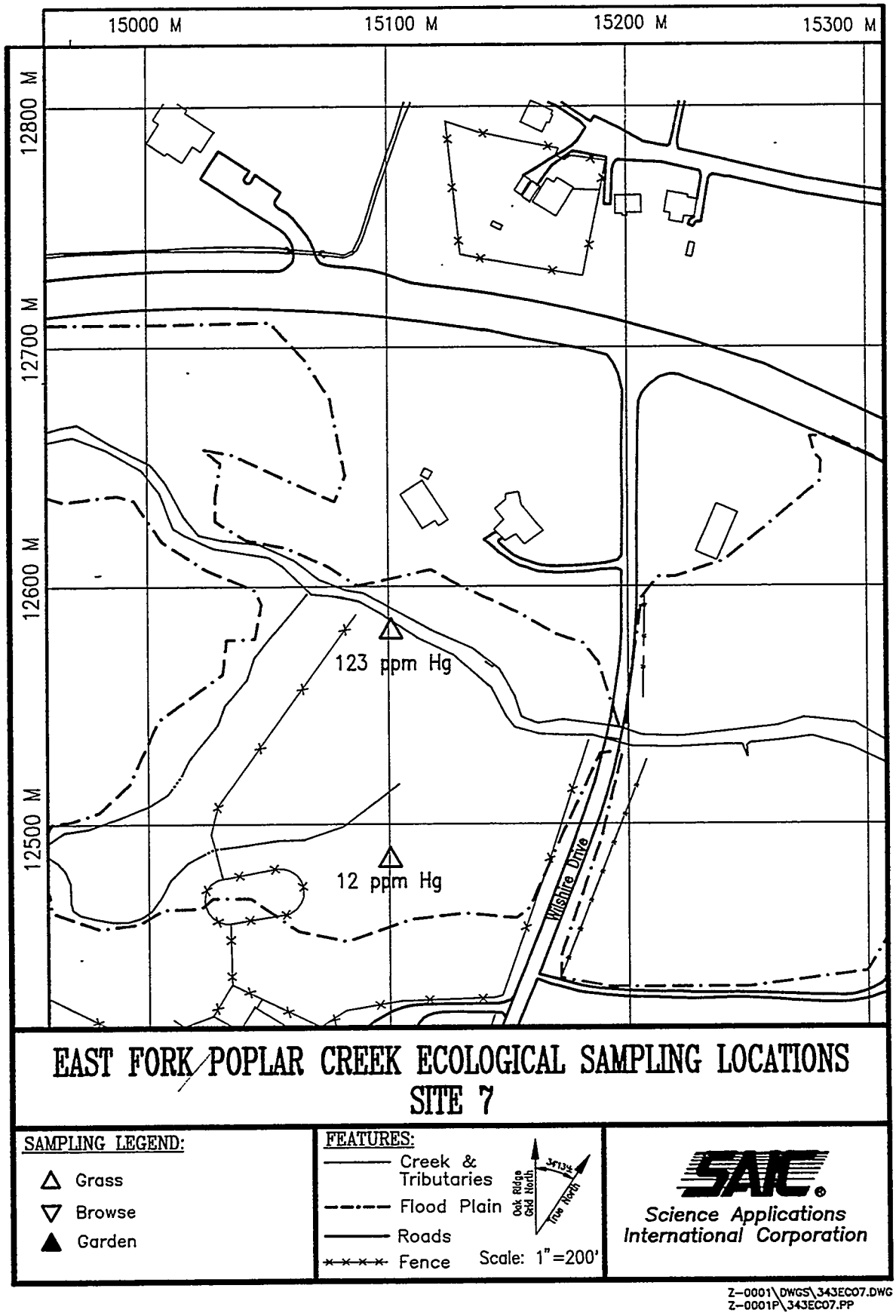


Fig. 6.12. EFPC sampling locations for grasses at Site 7.

point). At each site, samples of live and dead grass were taken at the creek edge and at 100 m (330 ft) from the creek. One sample of live grass was taken at Site 8 at the landowner's request. Browse (a mixture of vegetation typical of a deer's diet) was sampled at Site 3 at the creek edge and at 100 m (330 ft). Reference samples of grasses and browse were taken at the Hinds Creek reference site. Grasses and browse composites included adherent soil.

Beet roots, kale leaves, and tomato fruits grown in the EFPC floodplain at Site 3 were sampled and analyzed. The reference site for garden vegetation was a residential garden plot outside the EFPC floodplain. Beets and tomatoes were washed but not peeled, and kale leaves were not washed.

6.1.4.2 Environmental media

Air. The climate and air quality of EFPC, including meteorological and air monitoring data, are described in Sects. 2.3 and 3.2.6.

Groundwater. Information on groundwater is presented in Sect. 2.7, and existing groundwater data are found in Sect. 3.2.3.

Surface water. Information on the EFPC surface water system is found in Sect. 2.6, and surface water data are found in Sect. 3.2.1.

Soils. Soils at EFPC are described in Sect. 2.2, and soil chemical data are presented in Sect. 3.2.4.

Sediments. Information concerning the quality and contamination of EFPC sediments is presented in Sects. 2.6 and 3.2.2.

6.1.4.3 Indicator organisms

Studying every potential ecological receptor at EFPC would be virtually impossible. Instead, indicator organisms were chosen to represent selected groups of species occupying aquatic and terrestrial habitats (e.g., sediment, water column, and vegetation). Indicator organisms for the EFPC ERA were selected based on a process outlined in *Habitat Evaluation Procedures ESM 102* (FWS 1980). Subgroups of organisms within the aquatic and terrestrial communities were identified based on similarities in their mode of existence, diet, or taxonomic relationships (e.g., vegetation, benthic macroinvertebrates, birds). Indicator organisms were chosen for many subgroups constituting the EFPC ecosystem. Diagrammatic examples of the aquatic and terrestrial ecosystems at EFPC are illustrated in Figs. 6.14 and 6.15.

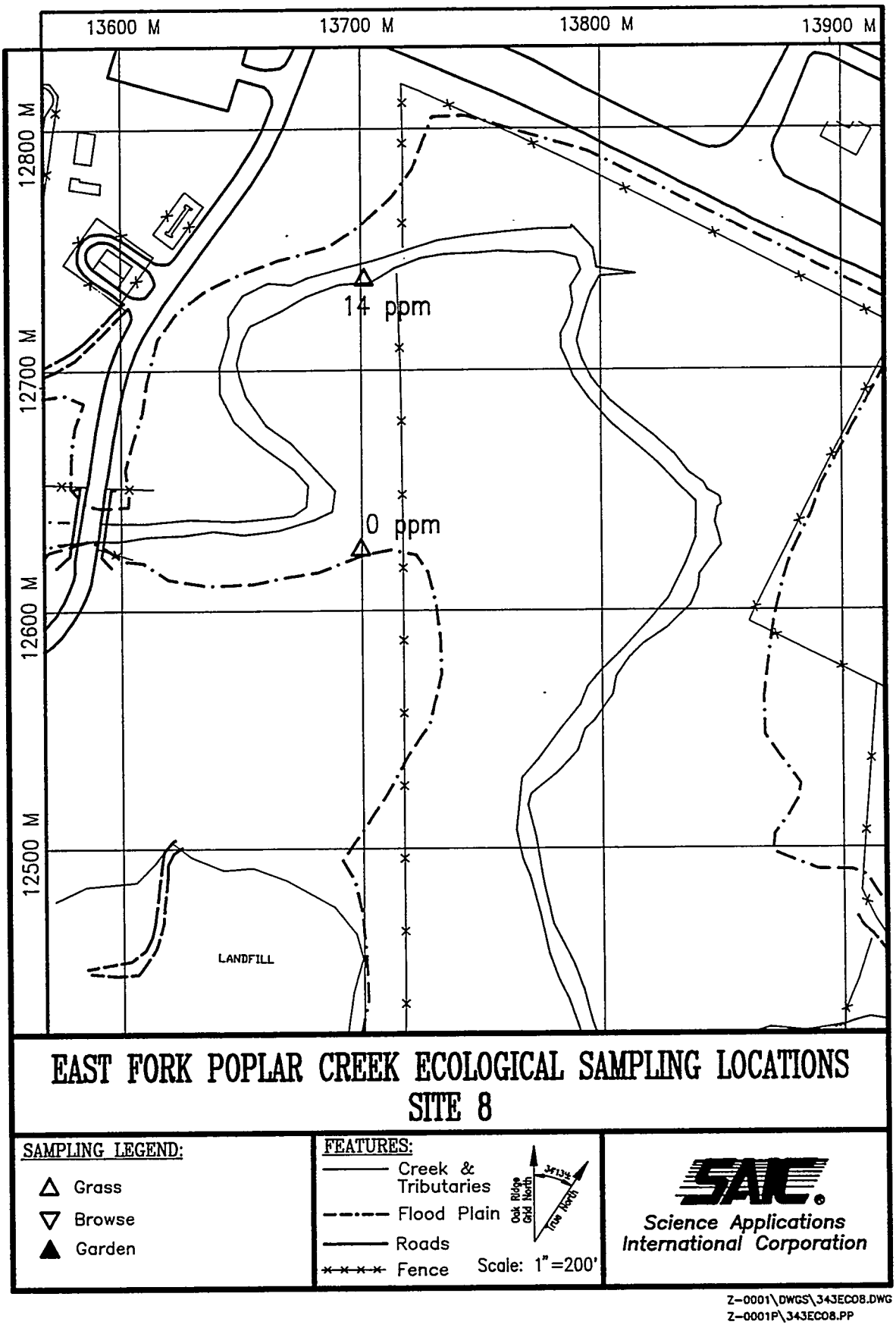


Fig. 6.13. EFPC sampling locations for grasses at Site 8.

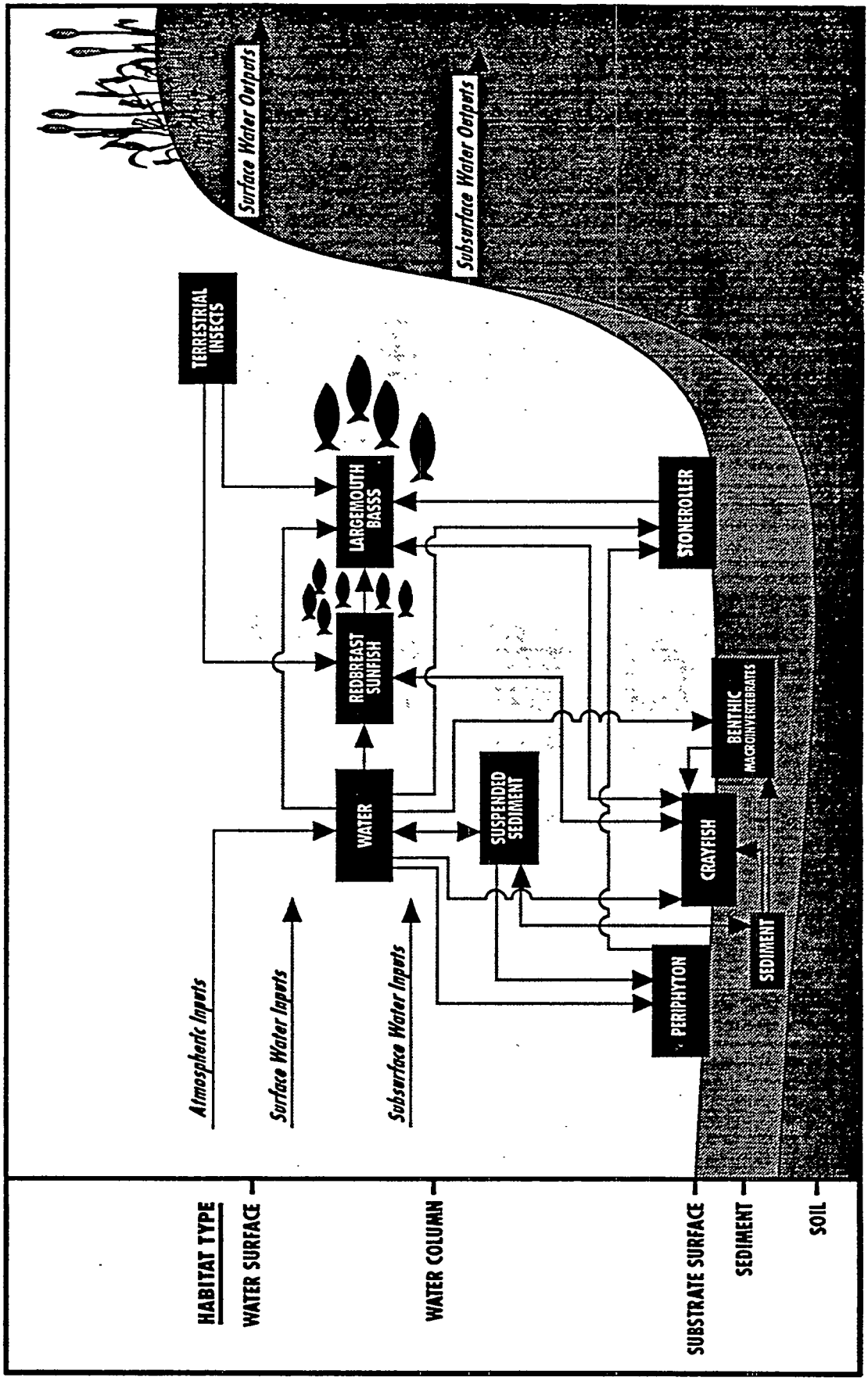


Fig. 6.14. Simplified food web for EFPC aquatic ecosystem.

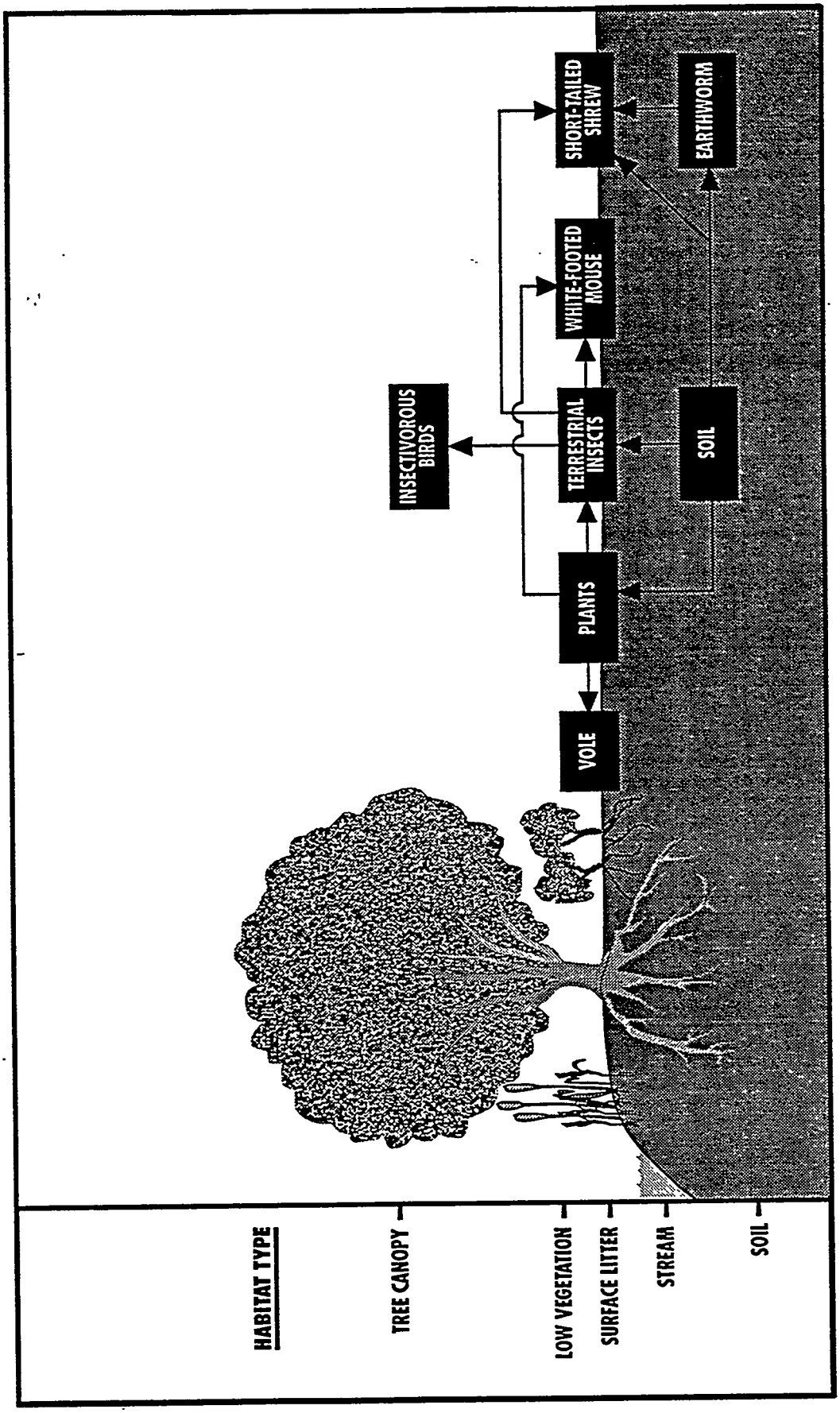


Fig. 6.15. Simplified food web for EFPC terrestrial ecosystem.

Indicator organisms were chosen to represent selected subgroups based on several considerations. They must occur in EFPC, be exposed to contaminated media, and have a limited home range so that they receive maximum exposure. Indicator organisms must be a key part of the food web. One or more indicator organisms need to represent the lower trophic levels in the food chain to allow modeling to be done up the food chain. It was further expected that the indicator organisms were found in adequate sample sizes and that measurable responses were obtainable. In many cases, threatened or endangered species, game species, and other species of special concern may not be suitable indicator organisms on these grounds. Other criteria for indicators are organisms that are vital to ecosystem energy and biogeochemical structure or function, are particularly sensitive to contaminants, or are specific bioaccumulators. An organism may be vital to the ecosystem if it is an essential prey species for an important predator. Sensitivity of an organism is typically determined by the rate of contaminant absorption from the environment, receptor tissue sensitivity and expression of toxic injury, and the rate of recovery from toxic injury or effect. Specific accumulators may have a normal level of sensitivity to a contaminant but may accumulate toxic compounds at an unusually high rate. Finally, indicator organisms need to be in harmony with natural resource trustee needs. The following subsection explains the selection of indicator organisms.

Aquatic community

A list of aquatic indicator organisms, their specific habitats, and measurement endpoints for each is found in Table 6.2.

Vertebrates. Fish are typical representatives of the aquatic vertebrates residing in the water column. Fish are important components of the aquatic ecosystem because they are consumers at several trophic levels, they are food sources to birds and mammals, and they have social and recreational value. Stonerollers (*Campostoma anomalum*), redbreast sunfish (*Lepomis auritus*), and largemouth bass (*Micropterus salmoides*) were the indicator organisms chosen to represent the water column vertebrates within EFPC and the Hinds Creek reference site. The stoneroller is an herbivorous fish that feeds exclusively on periphyton and thus represents "grazers" in the aquatic food web. The redbreast sunfish is a primary carnivorous fish, feeding upon smaller fish, terrestrial insects, and benthic invertebrates. The largemouth bass, although not particularly abundant in EFPC, represents the top predator fish species in the aquatic system.

Invertebrates (benthic macroinvertebrates and crayfish). Benthic macroinvertebrates perform several important functions, including mineralizing and recycling organic matter and serving as food for other organisms, especially fish (Lind 1979). Benthic macroinvertebrates that

Table 6.2. Aquatic indicator organisms and data to be collected for each

Indicators	Habitat	Measurement endpoints
Largemouth bass	Water column	Diversity, abundance, biomass, composite body burden
Redbreast sunfish		Diversity, abundance, biomass, composite and fillet body burden (2 sites only)
Other fish		Diversity, relative abundance, biomass
Stoneroller	Water column/substrate surface	Diversity, abundance, biomass, composite body burden
Benthic macroinvertebrates	Sediment	Diversity, relative abundance, composite body burden
Crayfish	Sediment/substrate surface	Relative Abundance, composite body burden
Periphyton	Substrate surface	Colonization rate, growth rate

live in or on sediment will have maximum potential for exposure to sediment porewater- and sediment-bound contaminants. As a result, they are susceptible to direct toxic effects from contaminants present in these media. In addition, these organisms may bioaccumulate contaminants and pass them up the food web as they are fed upon by predators. Crayfish and other benthic macroinvertebrates are found on the substrate surface living on cobble or in stream litter. Because they are omnivorous and scavenge on worms, dead fish, and plant debris, crayfish can be exposed to and can bioaccumulate contaminants.

Periphyton. Periphyton is a complex matrix of algae and heterotrophic microbes attached to submerged surfaces. Periphyton serves as a major food source for many stream invertebrates and herbivorous fish (Loar 1992). Periphyton represents the lower trophic level in the aquatic system. Since the biotic component of periphyton may replace itself in a matter of days, the periphyton community is useful for detecting short-term environmental changes. In addition, because the periphyton community is sessile and continually exposed to ambient stream conditions, rate changes and bioconcentration observed in periphyton are good indicators of instream toxicants.

Terrestrial community

A list of terrestrial indicator organisms, their specific habitats, and measurement endpoints for each is found in Table 6.3.

Vertebrates (small mammals). Using small mammals to monitor soil contaminants can be particularly effective if the species used are abundant, easily caught, do not migrate long distances, have a widespread distribution, and are generalists with respect to diet (Beardsley et al. 1978). Their relatively short life span (generally less than two years) facilitates long-term monitoring of contaminants. Because each generation reflects the present type and amount of contaminants in the environment, increases and decreases in contaminant concentration or the addition of new contaminants can be detected. The insectivorous shorttail shrew (*Blarina brevicauda*) is a good indicator organism because of its relatively high food intake. The shorttail shrew can bioaccumulate contaminants from its earthworm and insect prey at a fairly high rate. It also spends much of its time burrowing within the ground litter and just below the surface of the soil, where direct contact with any contaminants can occur. The white-footed mouse (*Peromyscus leucopus*) is an omnivore, and the vole (*Microtus pinetorium*) is an herbivore. White-footed mice spend most of their time on the soil and litter surface, while voles most often tunnel through the litter. These small mammals are prey for large predatory vertebrates.

Table 6.3. Terrestrial indicator organisms and data to be collected for each

Indicators	Habitat	Measurement endpoints
Small mammals insectivore herbivore omnivore	Floodplain ground surface	Diversity, relative abundance, age structure, habitat of occurrence, body burden
Birds		Diversity, relative abundance, habitat of occurrence,
Insects		Diversity, relative abundance, biomass, composite body burden
Plants		Diversity, dominance, % cover, recruitment
Small mammals	Floodplain surface soil, litter	Diversity, relative abundance, age structure, habitat of occurrence, body burden
Insects		Diversity, relative abundance, biomass, composite body burden
Birds	Floodplain brush and understory	Diversity, relative abundance, habitat of occurrence
Insects	Riparian	Diversity, relative abundance, biomass, composite body burden
Birds	Floodplain forest canopy	Diversity, relative abundance, habitat of occurrence
Insects		Diversity, relative abundance, biomass, composite body burden
Earthworms, insects	Subsurface soil	Relative abundance, composite body burden

Birds. Bird abundance and species richness are indicators of habitat quality. In addition, birds may be exposed to environmental contaminants, either directly or indirectly via the food web. Wrens feed on invertebrates, and bioaccumulation signals exposure and possible effects. Other birds are migrants and may be affected by contamination in the floodplain.

Invertebrates (earthworms and adult terrestrial insects). Invertebrates constitute a large portion of the terrestrial faunal biomass and are a major food source for numerous species of birds, reptiles, amphibians, and mammals. Earthworms and many immature forms of adult terrestrial insects are in intimate contact with the soil and interstitial water, providing maximum opportunity for exposure to contaminants.

Plants. The abundance and distribution of animal populations on the EFPC floodplain depends largely on the vegetation. Information on plant diversity, percentage of cover, dominant species, and recruitment by dominant species was gathered and compared to identical measures on the Mill Branch reference site. Plants are the lower trophic level and concentration measurements of browse and vegetables serves as input to these organisms that depend on plants for food.

Sampling design

The above representative organisms targeted for population surveys and sampling for body burden analysis were chosen to represent key exposure points in the EFPC ecosystem. Population surveys were done for terrestrial vegetation (Sect. 6.3.3.9) and birds (Sect. 6.3.3.6); both population surveys and body burden analyses were done for fish (Sects. 6.2.3.2 and 6.3.3.1), benthic macroinvertebrates (Sects. 6.2.3.2 and 6.3.3.2), terrestrial small mammals (Sects. 6.2.3.2 and 6.3.3.5), earthworms (Sects. 6.2.3.2 and 6.3.3.8), and flying insects (Sects. 6.2.3.2 and 6.3.3.7). Periphyton colonization rates were also determined (Sect. 6.3.3.3). Table 6.4 summarizes the surveys and analyses carried out for the EFPC ERA.

Sampling locations are described in Sect. 6.1.4.1. Table 6.5 shows the numbers of composite samples taken at each location. A detailed listing of sampling goals, sample numbers, and planned analyses, including QC samples, is presented in Appendix Q, Table Q.3.

The field investigations were conducted in the late summer of 1991 through the late summer of 1992. The aquatic ecosystem is considered to be most representative under conditions of stable base flow. The late summer and early fall were selected for intensive field surveys based on the need for the low rainfall conditions typically experienced during these seasons. Because of the present knowledge of the area, abundance of existing biological data on EFPC, and the schedule for the EFPC RI report, no initial field screening was performed.

Table 6.4. Measurements made for the EFPC ERA

Matrix sampled	Population measures			Contaminant concentration	Colonization rate	Growth rate
	Relative abundance	Biomass	Diversity			
Aquatic						
Fish	X	X	X	X		
Benthic macro-invertebrates	X	X	X	X		
Periphyton					X	X
Water				X		
Sediments				X		
Terrestrial						
Small mammals	X	X	X	X		
Birds	X		X	X		
Insects	X	X	X	X		
Earthworms	X		X	X		
Vegetation	X		X	X		
Soil				X		

Table 6.5. Numbers of ecological samples taken for the EFPC ERA

Sample type	Site 1	Site 2	Site 3	Site 4	Site 5	Site 6	Site 7	Site 8	Reference
Fish									
Stoneroller	2	2	2	2	1	1			2
Redbreast sunfish	2	1	2	2	2	1			1
Redbreast sunfish fillets			2			1			
Largemouth bass						1			
Benthic invertebrates									
Infaunal	2	1	1		1				1
Crayfish	2	2	2			1			2
Terrestrial invertebrates									
Earthworm	1	1	1	1	1	1			1
Insect, terrestrial origin	1	1	1	1	1	1			1
Insect, aquatic origin	1	1	1	1	1	1			1
Terrestrial small mammals									
Shrew				1		1			
Mouse	2	2	1	2	3				1
Vole				1					

Table 6.5 (continued)

Sample type	Site 1	Site 2	Site 3	Site 4	Site 5	Site 6	Site 7	Site 8	Reference
Birds									
Wren		1		1					
Heron					1				
Vegetation									
Fresh grass			3				2	3	3
Dead grass			2				2	3	1
Browse			2						1
TOTAL BIOLOGICAL 102	13	12	20	12	11	9	4	6	15
Surface water	1	1	1	1	1	1			1

Aquatic samples and surveys, as well as vegetation surveys, are expected to represent effects integrated over the lifetime of the individuals. Small mammal, earthworm, insect, and bird population surveys probably reflect seasonal influences as well as long-term impacts, and were interpreted in light of expected seasonal behavior of the species observed. The length of exposure represented by body burden analyses of small mammals, earthworms, and insects was interpreted by considering typical ranges and movements of these species.

Analytical Results

Results of body burden analyses are described in Sect. 6.2.3 and results of surveys are described in Sect. 6.3.3. Evaluation of data quality is described in Sect. 6.1.5.4. All measurements for biotic samples are wet weight; for abiotic (e.g., soil), the measurements are dry weight.

6.1.4.4 Physical stressor characteristics

Environmental changes induced by physical stressors and changes resulting from natural processes in the absence of remediation should be considered in the EFPC ERA. Minimum base flow and ambient water temperature regimes are two physical factors that are altered in EFPC due to operations at the Y-12 Plant. The minimum daily flow in EFPC is at least an order of magnitude greater than the minimum flow would be without contributions to stream flow from the Y-12 Plant and Oak Ridge Sewage Treatment Plant (Loar 1992). Furthermore, temporal variability in stream flow is very low in EFPC. In addition, ambient water temperatures in EFPC are higher and much less variable than in similar size reference streams such as Hinds Creek, especially close to the Y-12 Plant (and decrease longitudinally from the Y-12 Plant). Alterations in stream flow and temperature could result in reduced benthic species richness, and increases or decreases in total numbers and biomass.

Recovery from physical disturbances primarily takes place through the process of ecological succession.. Ecological succession (1) is the orderly and, therefore, often predictable process of community change, (2) results from the modification of the physical environment by the resident organisms, and (3) culminates in the establishment of an ecosystem that is as stable as is biologically possible (climax state) (Odum 1971). The EFPC floodplain includes areas undergoing various stages of ecological succession and areas in a presumed climax. The upper reaches contain channelized areas, and EFPC flows through commercial and residential developments. Middle reaches of the floodplain contain several large agricultural tracts, whereas the lower reaches, where the creek reenters ORR, contain relatively undisturbed flood-tolerant

bottomland hardwood forests. These forests, which are dominated by boxelder, are presumed to be the climax state, given the hydrological condition in the EFPC floodplain.

Ecological succession along EFPC depends on past, current, and future human activities and land management practices. These include mowing, grazing, fertilizing, burning, cutting, filling, dredging, and construction. Climatic cycles, storms, and severe fires can also have an effect on succession. If current land management practices and activities are discontinued, the human-maintained EFPC communities (e.g., pasture) would move toward the different and more forested stage of a climax state. The forest areas in the upper reach of the creek are already undergoing a shift in dominance as they recover from earlier changes. Ecological succession in the more heavily affected and managed areas along the middle reaches of the creek would also undergo shifts in dominance toward a climax state, but this would occur over a longer period of time (~200 years). The general pattern of ecological succession in upland areas outside the zone of frequently recurring floods would be one of transition from grassland to shrubs, followed by colonization by pine and eventual replacement of these by various shade-tolerant hardwood species.

6.1.5 Sources of Contamination

This subsection provides a brief overview of the types and sources of historical contamination. Following this, nature of the source data for this ERA are provided. Sources of background data and the nature of the data are presented next. Section 6.1.5 concludes with a brief mention of routes of exposure. Contaminants of concern are discussed in Sect. 6.1.5.5.

6.1.5.1 Historical data on chemical stressors

Chemical contaminant releases to EFPC have been discussed and summarized in Sect. 3.1.4. Sources of contaminants to EFPC include the Y-12 Plant and the Oak Ridge Sewage Treatment Plant wastewater treatment facility. Contaminant contributions from the Y-12 Plant are also introduced into the lower reaches of EFPC via Bear Creek. Several probable historical sources of other contaminants [such as polychlorinated biphenyls (PCBs) and polyaromatic hydrocarbons (PAHs)] are commercial facilities located within the EFPC floodplain; these have included automobile dealerships, service stations, and electrical substations.

Contaminants within the EFPC environment have been described and characterized by several previous studies (Gist 1987; Van Winkle et al. 1984; TVA 1985; Carmichael 1989; Loar 1992; Hinzman 1992; Appendix Q). An Oak Ridge Associated Universities (ORAU) study (Gist 1987), conducted between 1983 and 1987, included surveys for mercury in the surface soil along

Table 6.6. Target analytes, analytical methods, detection limits, and precision and recovery objectives for tissues

Analyte	Method	Target detection limits[a]	Relative % difference in lab duplicates	Percent spike recovery
METALS	NAA[b]	(μg analyte/g tissue)		
Antimony		0.3	35	90-110
Arsenic		2.0	35	90-110
Cadmium		4	35	90-110
Chromium		1	35	90-110
Mercury		0.2	35	90-110
Selenium		5	35	90-110
^{235}Uranium		0.1	35	90-110
^{238}Uranium		3.0	35	90-110
Zinc		60	35	90-110
ORGANICS				
Polynuclear Aromatic Hydrocarbons	8310[c]	(ng analyte/g tissue)		
Benzo(a)anthracene		1.5	40	
Benzo(a)pyrene		2.5	40	
Benzo(b)fluoranthene		2.0	40	
Benzo(g,h,i)perylene		8.0	40	
Benzo(k)fluroanthene		2.0	40	35-140
Chrysene		15	40	
Dibenzo(a,h)anthracene		3.0	40	35-140
Indeno(1,2,3-cd)pyrene		5.0	40	
Pyrene		30	40	
Pesticides	8080[c]			
Aldrin		1.0	25	30-135
2-4'-DDE		5.0	50	
4-4'-DDE		1.0	50	
2-4'-DDT		5.0	50	
4-4'-DDT		2.0	50	25-135
Dieldrin		1.0	40	30-135
Heptachlor		1.0	35	35-130
Heptachlor Epoxide		1.0	35	
Toxaphene		50.0	50	
Polychlorinated Biphenyls	8080[c]			
Aroclor-1254		20.0	50	
Aroclor-1260		20.0	50	25-135

[a]Detection limits for tissue are based on wet weight [1 g for neutron activation analysis (NAA), 10 g for organics].
[b]Tissues were homogenized before analysis by procedures described in SW-846 Method 3550 (EPA 1986a). Inorganics in tissue were analyzed by neutron activation (Radian 1993a, Appendix A).
[c]Tissue extracts for organics were analyzed in accordance with SW-846 methods (EPA 1986a).

Table 6.7. Target analytes, detection limits, and precision and spike recovery objectives for soils and vegetation

Analyte	Matrix	Target detection limits[a]	Relative % difference in lab duplicates	Percent spike recovery
METALS	Soil[b]	µg/g Soil		
Antimony		0.3	35	75 to 125
Arsenic		2.0	35	75 to 125
Cadmium		30	35	75 to 125
Chromium		20	35	75 to 125
Mercury		11	35	75 to 125
Selenium		30	35	75 to 125
Uranium		3.0	35	75 to 125
Zinc		120	35	75 to 125
	Vegetation[c]	ng/g Tissue		
Antimony		1	35	75 to 125
Arsenic		10	35	75 to 125
Cadmium		250	35	75 to 125
Chromium		50	35	75 to 125
Mercury		20	35	75 to 125
Selenium		60	35	75 to 125
Uranium		10	35	75 to 125
Zinc		200	35	75 to 125
RADIONUCLIDES[d]	Soil	pCi/g Soil		
^{60}Co		0.03	35	75 to 125
^{137}Cs		0.03	35	75 to 125
^{233}Pa		0.03	35	75 to 125
	Vegetation	pCi/Tissue		
^{60}Co		0.015	35	75 to 125
^{137}Cs		0.015	35	75 to 125
^{233}Pa		0.015	35	75 to 125

[a]Detection limits for tissue are based on wet weight (g).
[b]Soil was analyzed by NAA at the Analytical Chemistry Division, ORNL (Radian 1993a).
[c]Plant tissues were homogenized before analysis by procedures described in TP-309-9. Metals in plant tissues were analyzed by NAA at the Nuclear Services Center, North Carolina State University.
[d]Radionuclides were analyzed by ORAU, Oak Ridge, TN.

100-m (330-ft) transects across the floodplain from the Y-12 Plant to the Oak Ridge city limits in Roane county, grid sampling for mercury within a contaminated floodplain property, sampling for mercury from vertical profiles at various sites, and sampling of selected lawns and gardens in Oak Ridge. Sampling of vegetation, sewage sludge, groundwater, and two road-killed deer was performed for radionuclides, mercury, and other metals. The TVA study (1985), performed during 1984, sampled soils, sediments, surface water, fish, amphibians, crustaceans, and reptiles, which were then analyzed for mercury and other metals, radionuclides, organics, pesticides, and PCBs. The Y-12 Plant BMAP (Loar 1992; Hinzman 1992), begun in 1985, performs semiannual sampling of natural populations of fish which are then analyzed for mercury, PCBs, and cesium in fillet tissue. Ambient toxicity studies comprised of 7-d exposures of *Ceriodaphnia* and fathead minnows to water samples collected from EFPC have also been conducted semiannually by the Y-12 BMAP. Total residual chlorine concentrations are measured daily during the 7-d toxicity tests. Bioaccumulation studies of PAHs and PCBs have also been conducted annually using caged clams. The U.S. Geological Survey (USGS) conducted a survey of groundwater contamination by metals, radionuclides, and organics in 1987 (Carmichael 1989). This study sampled fourteen shallow wells at sites of known mercury contamination within the floodplain. Other one-time studies include a 1982 rapid assessment survey of mercury contamination in multiple media (i.e., sediments, mosses and liverworts, grasses, and fish) (Van Winkle et al. 1984) and the assessment of mercury and strontium in small mammals by Energy Systems (Talmage and Walton 1990).

6.1.5.2 Contaminant source data for risk assessment

EFPC RI Phase Ia sampling (1) addressed the nature and extent of contamination in the affected media and (2) screened for contaminants of concern. Media sampled included groundwater, surface water (both base flow and storm flow), soils, and sediments. Many of the primary sampling sites were areas historically shown to be contaminated; therefore, concentration values from Phase Ia used for calculations were likely to overestimate rather than to underestimate mean contamination levels in the floodplain. Contaminants of concern were identified using a toxicity concentration scoring system based on measured concentrations and established toxicity measures (EPA 1992b).

Phase Ib focused on determining the extent of contamination and on resolving data gaps in the historical and Phase Ia data. A tentative list of contaminants of concern for exposure of humans to water, soil, and sediments was prepared on the basis of results of Phase Ia sampling and analysis. Because biota are exposed through these media, those contaminants were included in the initial list of contaminants of concern for biota. Historical data on contaminants found in biological matrices by other investigators were used to supplement the list of contaminants of

concern for biota (Travis et al. 1989). These contaminants include several metals and a limited number of pesticides, PCBs, and PAHs. These compounds are listed in Table 6.6, along with the target detection limits. Other organic compounds routinely detected by the methods used were also reported; no tentatively identified compounds were reported.

Most historical sampling was restricted to the upper 10 to 15 cm (4 to 6 in.) of the soil profile, whereas exposure assessments can use contaminant concentration measurements from the top 50 cm (20 in.) of the soil. Limited data were available for much of the floodplain, and even fewer data were available from the lower reaches of the stream system. The historical sampling was not conducted using Level III or IV protocols. Quality assurance and quality control documentation was lacking, and analytical methods were not standardized [i.e., non-Contract Laboratory Program (CLP) methods]. Documentation for sample chain of custody and the location of sampling sites was not always maintained. Therefore, great reliance was placed on the Phase Ia and Ib data.

The list of target analytes for plants and garden vegetation was based on contaminants of concern for soil described in Sect. 5.1. However, several of these could not be measured in soil by the Analytical Chemistry Division of the Oak Ridge National Laboratory (ORNL) because of the constraints of the analytical conditions. These contaminants were dropped from the list of target analytes for vegetation because a major goal of vegetation analysis is to derive biotransfer factors from soil to plants, which cannot be done without results for soil. Because pesticides, PCBs, and PAHs are poorly soluble in water they are not expected to be transported extensively from soil into plants (Sect. 5.2.2.10; Travis and Arms 1988). The remaining contaminants of concern, along with antimony and selenium (which are routinely analyzed in soil), constitute the set of target analytes for garden produce and natural vegetation samples and are listed in Table 6.7, along with the target detection limits.

Phase Ia sampling of EFPC soils revealed the presence of low levels of ^{137}Cs, ^{60}Co, ^{234}U, ^{235}U, and ^{238}U. Total uranium was measured by neutron activation analysis (NAA). However, reference doses for low-level radiotoxicity to individuals of threatened and endangered species and to populations typical of the EFPC ecosystem are not currently available. For this reason and because of the limited amounts of tissue expected from the ecological sampling, other radionuclides were not determined in samples of aquatic and terrestrial animals. Because benchmark values for exposure of plants and animals to radionuclides do not exist, data on concentrations of radionuclides would not contribute meaningfully to risk assessment for these receptors. In addition, surveys of deer killed during managed hunts on the ORR showed that very few deer exceeded the Tennessee Wildlife Resources Agency confiscation limits of 30 pCi

^{90}Sr/g bone and 5 pCi ^{137}Cs/g muscle or liver (Evans 1992). Therefore, radionuclides were not measured in natural vegetation samples. Radionuclides of concern in crops for human consumption are those detected at elevated levels in soils during Phase Ia sampling and analysis. They are listed in Table 6.7.

6.1.5.3 Background or reference data

A site on Hinds Creek near Norris, Tennessee (Fig. 6.9), was used to provide comparative chemical concentration data for soils, sediments, surface water, and selected biota (i.e., fish, periphyton, benthos, and insects). A site on Mill Branch (Fig.6.10), a tributary of EFPC, was used to provide comparative chemical data for selected biota only (i.e., birds, small mammals, and earthworms).

The BMAP has used a similar stream system as a reference—Brushy Fork, immediately north of Oak Ridge. This stream was not selected for use as a reference stream for the EFPC RI because of high levels of certain contaminants, notably PCBs. BMAP also uses Hinds Creek as a reference.

6.1.5.4 Nature of the source data

Phase Ia sampling was performed using a sampling and analysis plan entitled *Environmental Restoration Program East Fork Poplar Creek-Sewer Line Beltway Integrated RCRA/CERCLA/NEPA Assessment Phase Ia, Parts I & II* (LWA 1991). All Phase Ia data used were sampled and analyzed following a consistent set of procedures, and the quality of the analytical data was documented under EPA Level IV protocols.

Phase Ib data were collected and analyzed using *Environmental Restoration Program East Fork Poplar Creek-Sewer Line Beltway Integrated RCRA/CERCLA/NEPA Phase Ib Sampling and Analysis Plan for Soil, Sediment, and Water* (Radian 1992). This plan provided for quality assurance controls and data validation during sampling and analysis.

Precision, accuracy, representativeness, completeness, and comparability (PARCC) parameters

PARCC parameters were used to evaluate the quality of data obtained from ecological sampling. These parameters are discussed in the following subsections.

The precision of analyses for organic analytes was evaluated from data on spiked samples, as small sample sizes precluded preparation of laboratory duplicates of field samples. Samples

evaluated for precision and accuracy of inorganic analyses included laboratory duplicates of field samples, spiked samples, and duplicate analyses of laboratory standards. Control charts and details of precision and accuracy evaluations are presented in Appendix K. Results of precision and accuracy evaluations are summarized in Table 6.8.

Precision. Precision measures the reproducibility of measurements under constant conditions. Analytical precision was evaluated by comparing the relative percent difference (RPD) between duplicate analyses of single samples to criteria established in the sampling and analysis plans (SAPs) (Radian 1993a, 1993b). Of the target analytes, the specified upper limits for RPDs were 40% for pyrene and 35% for aldrin, dieldrin, and heptachlor (Radian 1992b). RPDs of 35% were specified for inorganic analytes (Radian 1993b). Precision objectives for organic analytes were met by 93.6% of samples. Precision objectives for inorganic analyses were met for 93% of vegetation samples and for 97% of faunal samples. Appendix K presents control charts for RPDs of analyses of inorganic analytes in vegetation (Fig. K.1) and fauna (Fig. K.2) and for organic analytes in fauna (Fig. K.3). Recovery and RPDs of laboratory control samples for analysis of PAH analytes are presented in Appendix K, Table K.1, and recovery and RPDs of laboratory control samples for analysis of pesticide/PCB analytes are presented in Table K.2.

No criteria for sampling precision were established in the SAPs for biota. However, sampling precision can be measured by comparing collocated samples of the same medium. Many of the biota samples analyzed comprised many individuals composited and homogenized into a single sample. This procedure was adopted to ensure sufficient sample size for analysis of small organisms and to reduce the potential for analytical variability because of differences between individual organisms. However, individual organisms were used in the analysis of redbreast sunfish fillets, bass, mice, shrews, vole, wrens, and heron.

To evaluate the potential uncertainty introduced by using individual animals rather than composite samples, a comparison was made of the intersample variabilities of mouse samples, which were individual animals, crayfish composites, which contained several individuals, and stoneroller composites, which were made up of many individuals. Whenever samples of a single taxon could be paired at a given site, the RPD between results for each analyte in that taxon was calculated. Results reported below detection limits were not used in this evaluation. The mean RPD for mouse samples was 45.3% and the maximum was 182%. In contrast, the mean RPD for crayfish composites was 90.3% and the maximum was 198%, whereas the mean RPD for stoneroller composites was 40% and the maximum was 192%.

Table 6.8. Precision and accuracy summary for EFPC biota analyses

Parameter	Measure	Analyte class	Criterion	Fraction meeting criterion
Precision	RPD	Organic	35% or 40%	29/31
	RPD	Inorganic		
		vegetation	35%	52/56
		fauna	35%	179/185
Accuracy	Spike recovery	Organic	30-140%	121/124
	Spike recovery	Inorganic		
		vegetation	75-125%	59/60
		fauna	75-125%	47/47
	Reference standards	Inorganic		
		vegetation	±10%	62/63
		fauna	±10%	66/67

In many cases, the values reported for an analyte at each site were all below detection limits. However, at two site, RPDs for mice, crayfish, and stonerollers could be calculated for a total of 18 analytes (Table 6.9). In 12 cases, RPDs for crayfish composites were higher than for mouse samples, whereas in 9 cases, each RPD for stoneroller composites was higher than each RPD for stoneroller and mouse samples. Therefore, although crayfish composites were more variable than composites comprising many stonerollers, and intersample variability between individual mice at a single sample location was not markedly different from composites of large numbers of stonerollers.

Accuracy. Analytical accuracy was assessed by evaluating matrix spike recovery of organic and inorganic analytes and by comparing analytical results with known concentrations of inorganic analytes in standard reference materials. Standard reference materials for organic analytes in biological tissues were not available. Recovery and RPDs of matrix spike samples for analysis of PAH analytes are presented in Appendix K, Table K.3, and recovery and RPDs of matrix spike samples for analysis of pesticide/PCB analytes are presented in Table K.4.

Accuracy objectives established in the SAPs for biota were matrix spike recoveries ranging from 30 to 140% for organic analytes (Radian 1993a) and 75 to 125% for inorganic analytes (Radian 1993b). The SAP for inorganic analytes (Radian 1993b) also specified that results should agree within 10% of the stated concentrations of standard reference materials for inorganic samples. These criteria were met by 98% of matrix spike samples for organic analytes, 98% of vegetation matrix spike samples for inorganic analytes, 100% of fauna matrix spike samples for inorganic analytes, and 98.5% of laboratory control standards for inorganic analytes.

Representativeness. Representativeness describes the degree to which sample data reflect the population sampled. In the EFPC RI, biotic sampling was biased because historical data had indicated that contaminant concentrations in biota were location-specific and because the large size and complex nature of the EFPC floodplain and creek made it impractical to develop an unbiased or random sampling plan.

The rationale for choosing sampling sites was presented in the SAP for ecological risk assessment (Radian 1993a) and also in Sect. 6.1.4.1. Sample sites were chosen (1) to represent areas believed to receive high immediate impact from the Y-12 Plant or high contaminant deposition or (2) to determine potential effects of discharges from the Oak Ridge Sewage Treatment plant from Bear Creek. Six sampling areas were selected along EFPC. Specific sampling locations within each area were chosen for each type of biota, the indicator organism sampling locations overlapping as much as was feasible. Reference areas were selected that

Table 6.9. Comparison of RPDs for inorganic analytes in mice, crayfish, and stoneroller field duplicate samples

Site	Analyte	RPD		
		Mouse	Crayfish	Stoneroller
1	Chromium	4.0	20.5	15.6
	Selenium	18.6	0.8	7.6
	Zinc	1.9	8.8	3.3
	Anthracene	12.3	4.6	8.0
	Benzo[a]pyrene	10.5	12.8	1.1
	Benzo[k]fluoranthene	13.0	8.0	0.0
	Aroclor 1260	2.9	7.7	1.2
	DDE	0.8	5.8	1.3
	Heptachlor epoxide	6.7	22.4	1.7
2	Chromium	11.3	6.2	1.8
	Mercury	12.0	6.0	15.7
	Zinc	1.0	0.9	1.1
	Anthracene	11.2	15.6	15.1
	Benzo[a]pyrene	5.3	35.0	10.0
	Benzo[k]fluoranthene	4.0	39.8	11.1
	Naphthalene	0.6	27.6	45.2
	Aroclor 1260	7.3	9.6	3.7
	Heptachlor epoxide	13.7	38.1	1.4

represent as closely as possible the various habitats found at EFPC. The reference site for aquatic and flying insect investigations was on Hinds Creek, which has been used for several years as a reference stream by investigators at ORNL. The reference area for avian, plant, small mammal, and earthworm investigations was located on Mill Branch, a tributary of EFPC.

Completeness. Completeness is assessed by determining the percentage of measurements that resulted in valid data. The SAPs for biotic sampling established target numbers of samples of each taxon and at each site. The fractions of planned vegetation samples that were actually taken are summarized in Appendix K, Table K.5, and the fractions of planned faunal samples and numbers of samples submitted for analysis of semivolatile organics, pesticides/PCBs, and inorganics are summarized in Table K.6.

Although target detection limits for inorganic analytes in some samples were not met because of interferences, none of the analytical data were rejected for reasons of quality of analysis because the analyses were categorized as special analytical services (Level V), and detection limits were presented as targets rather than requirements. Similarly, a small fraction of the analyses of organic analytes failed to meet target detection limits but were retained as qualified data for the ERA.

Comparability. Comparability of results might be compromised by variability introduced by the use of different sizes of samples from biotic populations. At the extremes, samples of some taxa (e.g., shorttail shrew, vole) consisted of single organisms, whereas samples of flying insects comprised a few hundred individuals, and stoneroller composites contained up to nearly 100 individuals. Evaluation of variability between field duplicates (Table 6.9) showed that variability between individual mice was not markedly different from variability between stoneroller composite samples. Therefore, sample size was judged not to have compromised the comparability of analytical results.

Historical data on body burdens of mercury in redbreast sunfish are discussed in Sect. 6.2.3.1 and are compared to results of RI sampling in Sect. 6.2.3.3. Historical data on contaminants in EFPC fish are not directly comparable to most of the RI data because the analyses reported previously were done on fillets (because they were intended for human health risk assessment). A comparison of fillets and whole-body composite samples demonstrated that mercury levels in fillets and whole-body composites of redbreast sunfish were comparable, whereas PCB levels in whole-body composites were higher than in fillets.

Mercury concentrations at Site 3 were 2145 μg/kg in fillets and 1863 μg/kg in whole-body composites, for a ratio of 1.15 in fillets compared to whole-body composites, and at Site 5 the

concentrations were 722 μg/kg in fillets and 476 μg/kg in whole-body composites, for a ratio of 1.51. Therefore, mercury levels in fillets appear to be slightly higher than in whole-body composites in the current samples.

Analytical results for whole-body composites and fillets of redbreast sunfish sampled at Sites 3 and 5 were compared. The Aroclor 1260 concentrations at Site 3 were 615 μg/kg in fillets and 2800 μg/kg in whole-body composites (averages of 2 samples each), for a ratio of 0.22 in fillets compared to whole body. At Site 5, the concentrations were 170 μg/kg (single fillet) and 990 μg/kg (average of 2 whole-body composites), for a ratio of 0.17. Therefore, PCB levels in fillets were consistently below the levels in whole-body composites at the corresponding sample locations by a factor of ~5. This observation is consistent with the tendency of PCBs to accumulate in body fat and organs rather than in muscle tissue.

6.1.5.5 Evaluation of ecological contaminants of concern

Procedures for evaluation of ecological COCs followed recommendations of EPA for evaluation of analytical data (EPA 1989c). The criteria used and the results of the evaluations are described in the subsections below. Summary data on analytes found in different biotic media at reference sites and at the EFPC sampling sites are presented in Table Q.2 (Appendix Q). Analytes were not listed in Table 6.6 and 6.7 if they were not identified in either the background samples or the EFPC samples. Table 6.10 presents a summary of the evaluation of COCs. In this table, the analytes are listed and there are entries to indicate whether they were retained or rejected as COCs in each of the indicator organism groups. If an analyte was rejected, the reason for its rejection is also indicated.

Frequency of occurrence

Analytes are typically included as COCs only if they are present in at least 5% of samples (1 in 20 samples or at least one of <20 samples) of the same medium (EPA 1989c). Because the number of EFPC samples of each type of biota was small, any analyte positively identified in at least one sample qualified that analyte as a COC for that organism. In some cases analytes were not detected in EFPC samples but were identified in samples from the reference area. These analytes were assumed to be present at low concentrations in the EFPC samples and were included as COCs (EPA 1989c). Concentrations of contaminants that were considered to be present in site samples but were reported below detection limits in specific samples were given proxy concentrations of half the detection limit when calculating average concentrations.

Table 6.10. Summary of COC selection for biota

	Detection limit[a] (ng/g sample)	Stoneroller	Redbreast	Bass	Crayfish	Benthic invert.	Aquatic insect	Terrestrial insect	Earthworm	Mouse	Shrew	Vole	Wren	Heron liver	Heron feather
PAHs															
Acenaphthene	79	+	+	b	+	c	b	c	b	c	+	+	+	c	b
Acenaphthylene	106	+	d	b	+	+	b	+	b	d	+	+	c	+	b
Anthracene	29	d	+	b	+	+	b	d	b	d	+	+	+	+	b
Benzo(a)anthracene	0.73	+	e	b	+	+	b	+	b	d	+	+	+	c	b
Benzo(a)pyrene	1.3	+	c	b	+	+	b	+	b	d	+	+	+	+	b
Benzo(b)fluoranthene	0.90	+	e	b	+	+	b	+	b	d	+	+	+	c	b
Benzo(g,h,i)perylene	2.4	+	c	b	+	+	b	d	b	c	+	+	c	c	b
Benzo(k)fluoranthene	0.62	+	e	b	+	+	b	+	b	d	+	+	+	+	b
Chrysene	6.0	+	c	b	+	+	b	d	b	c	+	+	c	c	b
Dibenzo(a,h)anthracene	0.90	+	+	b	+	+	b	+	b	c	+	+	c	c	b
Fluoranthene	10	+	c	b	+	+	b	d	b	d	+	+	c	c	b
Fluorene	11	+	d	b	d	+	b	d	b	d	+	c	c	c	b
Indeno(1,2,3-cd)pyrene	2.7	+	c	b	+	+	b	+	b	d	+	+	c	+	b
Naphthalene	1.0	+	d	b	d	+	b	+	b	+	+	+	+	+	b
Phenanthrene	31	+	c	b	+	+	b	d	b	d	+	+	c	c	b
Pyrene	12	+	d	b	+	+	b	d	b	c	+	c	c	c	b
Pesticides															
Aldrin	2.1	+	c	b	+	+	b	d	b	d	+	c	c	+	b

Table 6.10. (continued)

	Detection limit[a] (ng/30 g sample)	Stoneroller	Redbreast	Bass	Crayfish	Baultic invert.	Aquatic insect	Terrestrial insect	Earthworm	Mouse	Shrew	Vole	Wren	Heron liver	Heron feather
Pesticides (cont'd)															
α-BHC	5.3	+	+	b	+	+	b	+	b	+	c	+	c	+	b
ß-BHC	18	+	+	b	c	+	b	d	b	+	c	+	c	c	b
γ-BHC	2.3	+	+	b	+	+	b	d	b	+	+	+	c	c	b
δ-BHC	5.0	c	c	b	c	+	b	+	b	d	+	+	c	c	b
α-Chlordane	5.6	+	+	b	+	c	b	d	b	d	+	+	+	+	b
γ-Chlordane	4.7	+	+	b	+	c	b	+	b	+	c	c	c	c	b
pp'-DDD	7.4	+	+	b	c	c	b	c	b	c	c	c	c	c	b
pp'-DDE	9.5	+	+	b	c	+	b	c	b	+	+	+	+	c	b
pp'-DDT	4.7	+	+	b	c	c	b	c	b	d	c	+	+	c	b
Dieldrin	13	+	+	b	+	+	b	+	b	d	+	+	c	+	b
α-Endosulfan	4.5	c	c	b	c	c	b	c	b	c	c	c	c	c	b
ß-Endosulfan	5.8	c	c	b	c	c	b	d	b	+	c	c	c	c	b
Endosulfan sulfate	6.2	+	c	b	c	c	b	+	b	c	c	c	c	c	b
Endrin	82	+	c	b	c	c	b	c	b	c	c	c	c	c	b
Endrin aldehyde	2.0	+	c	b	+	c	b	d	b	c	c	c	c	c	b
Endrin ketone	5.3	c	+	b	+	c	b	+	b	c	c	c	c	c	b
Heptachlor	5.3	+	+	b	c	c	b	d	b	c	c	c	c	c	b
Heptachlor epoxide	50	+	c	b	+	+	b	d	b	+	c	c	+	+	b

Table 6.10. (continued)

	Detection limit[a] (ng/g sample)	Stoneroller	Redbreast	Bass	Crayfish	Benthic invert.	Aquatic insect	Terrestrial insect	Earthworm	Mouse	Shrew	Vole	Wren	Heron liver	Heron feather
Pesticides (cont'd)															
Methoxychlor	50	c	c	b	c	c	b	c	b	c	c	c	c	c	b
Toxaphene	50	c	c	b	c	c	b	c	b	c	c	c	c	c	b
PCBs															
Aroclor 1016	20	c	+	b	+	c	b	+	b	c	c	+	c	c	b
Aroclor 1232	20	c	c	b	c	c	b	c	b	c	c	c	c	c	b
Aroclor 1248	10	c	c	b	c	c	b	c	b	c	c	c	c	c	b
Aroclor 1254	4	c	c	b	c	c	b	c	b	c	c	c	c	c	b
Aroclor 1260	6.8	+	+	b	+	+	b	+	b	+	+	+	+	+	b
Inorganics															
Antimony	1	+	d	c	c	d	d	+	d	+	+	+	+	c	c
Arsenic	10	d	c	c	c	d	d	+	d	c	+	c	c	c	c
Cadmium	250	c	c	c	+	+	d	c	+	d	+	c	c	c	c
Chromium	50	+	d	c	d	+	+	+	d	+	+	+	+	c	+
Mercury	20	+	+	+	+	+	+	+	+	+	+	c	+	+	+
Selenium	60	+	d	+	+	+	+	+	+	+	+	c	+	+	c
Uranium	200	+	c	c	+	+	c	c	+	c	c	c	c	c	c
Zinc	10	+	d	+	+	+	d	d	+	d	+	+	+	+	+

[a]Method detection limits, expressed as ng/g tissue.

[b]Not analyzed

[c]Not detected.

[d]Identified at or below reference criteria.

[e]Less than 5x blank values.

"+" Denotes contaminants of concern.

The detection frequencies of analytes in biota are summarized in Table Q.2. By the criterion of frequency of occurrence, all PCBs except Aroclor 1016 and Aroclor 1260 were eliminated as COCs (Table 6.10, indicated by note c). All of the 16 PAHs were retained as COCs in both aquatic and terrestrial biota. Of 21 pesticides, three were eliminated as COCs in aquatic biota and five were eliminated as COCs in terrestrial biota. All inorganic analytes were retained as COCs, although among terrestrial biota uranium was identified only in earthworms.

Comparison of detection limits to toxicity criteria

Analytes whose toxicity criteria are below detection limits are to be retained as COCs even if they are not positively detected, as long as there is reason to believe they may be present at the site. None of the toxicity criteria for tissues (see Sect. 6.4.1.1 for further discussion) were below detection limits, so none of the undetected analytes were retained as COCs for this reason.

Presence in blanks

If analytes are detected in blanks, there is a possibility that contamination of samples has also occurred. Therefore, the presence of analytes in blanks must be evaluated. Results are flagged in the data report for each analyte that is found in the associated blank. For certain analytes that are typical laboratory contaminants, the concentrations in samples must be 10 times the concentrations identified in blanks in order for the sample values to be accepted as valid. However, these analytes are predominantly volatile organics, which were not analyzed in the biotic samples. For other analytes, the concentrations observed in samples must be at least 5 times the concentrations detected in blanks. If all results for a given analyte are <5 times the value found in the associated blank, that analyte is excluded as a COC. Otherwise, the results are treated as non-detects, which for the EFPC RI means that they were assigned a proxy value of one-half the detection limit.

Blanks were analyzed with each sample group of EFPC biota samples. The analytical data were evaluated, and samples whose associated blanks showed evidence of contaminated blanks are indicated in Table 6.10 (Note e). Organic analytes detected in blanks included benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, and chrysene in blanks associated with three samples of redbreast sunfish. Inorganic analytes detected in blanks included antimony and cadmium in blanks associated with vegetation. A total of eight analytical results were changed to proxy values because of their detection in blanks.

Comparison to background

Concentrations of contaminants at the site must be greater than at a reference area not impacted by the site. When there were two or more samples from the reference site, the 95% upper confidence limit was used for comparison with sample results. However, in most cases, there were insufficient reference site samples to calculate a confidence interval for comparison with site data. To allow for uncertainty in the range of reference data in these cases, two times the single observed value (the full value of the reported detection limit) was used for the reference value. Analytes rejected as COCs because they were less than or equal to background values are indicated in Table 6.10 by Note d.

Evaluation of tentatively identified compounds

EPA guidance (1989c) for evaluation of tentatively identified compounds (TICs) calls for the thirty most abundant chromatographic peaks not on the target analyte list to be identified, if possible, by comparison to a library of analytical results for other organic compounds. Risks from TICs are to be evaluated only if it appears that they would dominate the risks to receptors. No explicit guidance on evaluation of TICs in ecological risk assessment was presented by EPA (EPA 1992a). TICs were excluded from this risk characterization because it was expected that the complexity of the biological matrices would result in many TICs derived from the tissues themselves, for which reference analytical results are not available. Therefore, in most cases it would not be possible to distinguish between degradation products of organic contaminants and the many organic molecules produced by the biota being sampled.

6.1.5.6 Routes of exposure for indicator organisms

Two major exposure sources exist within EFPC and its floodplain. Water effluents from the Y-12 Plant continue to expose aquatic indicator organisms during normal flow especially in the upper part of EFPC below Lake Reality and can contribute to the exposure of terrestrial organisms during floods through direct contact or through deposition. The previously contaminated floodplain soil also contributes to the exposure of terrestrial indicator organisms through dermal contact and ingestion, and to aquatic organisms through erosion or scouring and redeposition. Direct exposure routes could exist through the air, shallow groundwater, surface water, instream sediments, and soil. Indirect exposure occurs through ingestion of contaminated forage or prey. The next major section deals with the findings for exposure.

6.2 EXPOSURE ASSESSMENT

According to the *Framework for Ecological Risk Assessment* (EPA 1992a), the ERA process consists of four steps—problem formulation, exposure characterization, effects characterization, and risk characterization. Section 6.2 deals with exposure characterization, which describes the abiotic and biotic environmental attributes, along with the route, magnitude, frequency, duration, trend, and spatial pattern of exposure of each indicator population or habitat component in relation to a chemical or physical stressor. The four-step process is described more fully in Sect. 6.1.2.

6.2.1 Characterization of the Exposure Environment

Exposure environments in EFPC and its floodplain consist of a complex mixture of flow regimes, streambed conditions, and terrestrial habitats varying from commercial through agricultural to woodland. Surface water is the primary medium of exposure for aquatic organisms to contaminants currently being released to EFPC via surface water from the Y-12 Plant and to contaminants in floodplain soils that are transported via erosion, surface runoff, and shallow groundwater. Soil, via food chains, is the primary medium of exposure for terrestrial biota. Stream sediments are a second major exposure route for benthic macroinvertebrates and for terrestrial consumers by way of flood deposits on vegetation. In both aquatic and terrestrial communities, indirect exposure of organisms via the food web is expected to be important. Methylation/ demethylation of mercury in sediments and stream water and biodegradation of organic contaminants are the most important chemical transformations. The following subsections discuss the relationships between contaminant sources and the biotic indicator exposed by different routes.

6.2.1.1 Relationship of contaminant sources and endpoint biota (indicator organisms)

The conceptual model of releases from the Y-12 Plant to EFPC is shown in Fig. 6.16. This figure shows that contaminants are released from the Y-12 Plant into EFPC, which transports them into the floodplain ecosystem. Biota may be exposed to contaminants in surface water and sediments, in floodplain soils, in shallow groundwater, and in their food.

Floodplain soils contain contaminants deposited there by EFPC over many years. These soils are the primary sources of contaminants, but exposures occur in secondary source media such as surface water. The relationships of sources to exposure media are discussed in the following subsections.

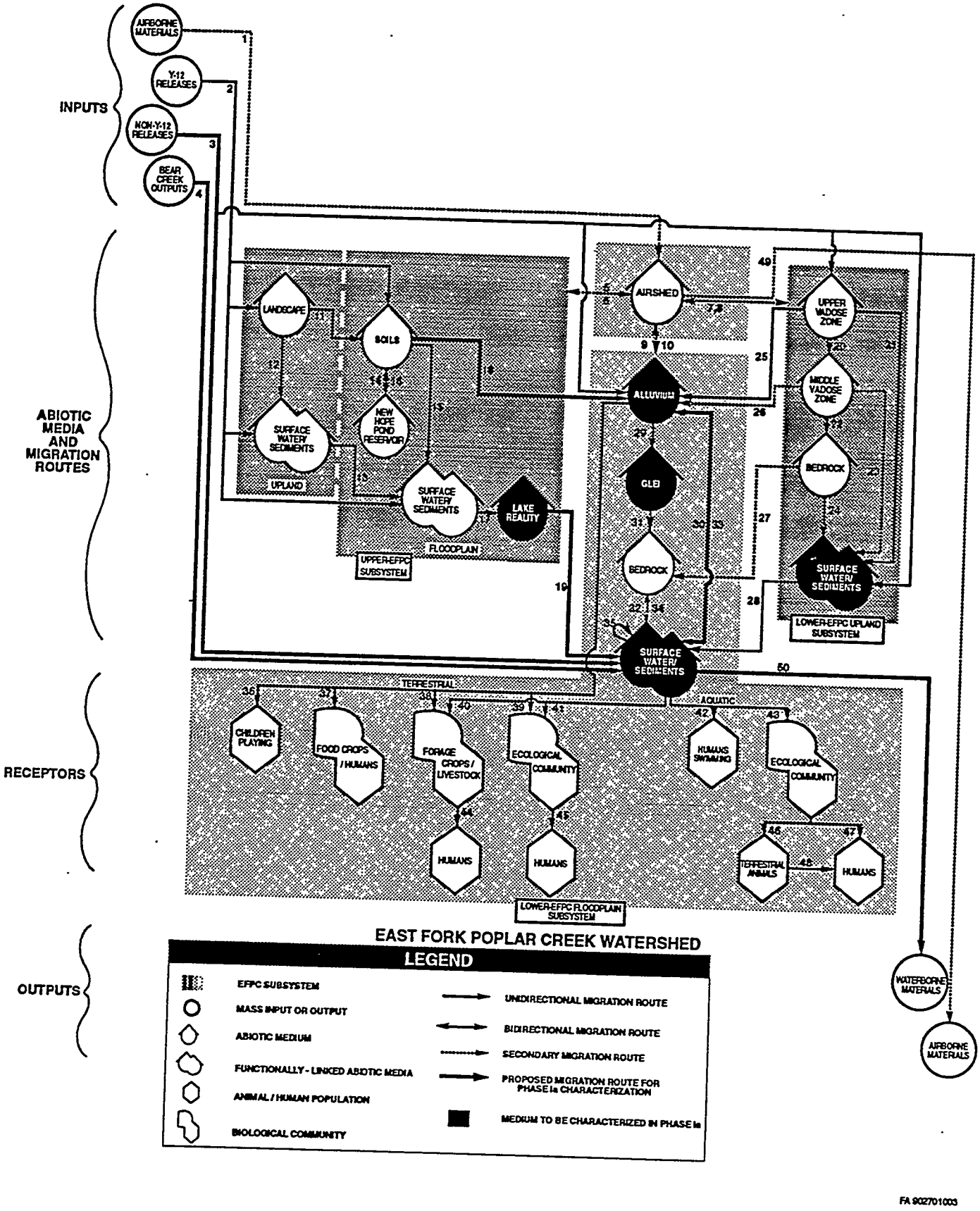


Fig. 6.16. Conceptual model of releases from the Y-12 Plant to EFPC.

Surface water. Surface water is a highly mobile, transient medium containing a mixture of soluble and suspended contaminants from the Y-12 Plant and a variety of point and nonpoint sources outside the Y-12 Plant. Because EFPC originates within the Y-12 Plant, the relative contribution of offsite sources is presumed to be small at the sampling site at Pine Ridge Gap and to increase with distance downstream. During much of the year, several of the tributaries to EFPC have little or no flow in the absence of industrial or commercial discharges or storm events. Several other tributaries make substantial contributions to the flow and suspended solids load in the middle portion of EFPC. The most important of these is probably the tributary labeled CC in Map 4, which drains an area of ~450 ha (1200 acres) north and east of coordinates 38500N and 60000E (roughly, at the intersection of Illinois Avenue and Oak Ridge Turnpike). Mill Branch, entering EFPC at ~48500E, is also a significant contributor of runoff and sediments from residential and undeveloped areas; Bear Creek, entering EFPC at ~22700E, contributes potentially contaminated runoff from the west end of the Y-12 Plant.

Surface water is a medium of exposure for fish, crayfish, aquatic insects, benthic macroinvertebrates, and periphyton. It can also expose terrestrial mammals and insects and birds that drink from creeks or eat aquatic biota. During floods, terrestrial plants can also be exposed to contaminants in surface water by contact with dissolved contaminants and by deposition of contaminated suspended sediments. Terrestrial animals can be exposed via contact with an ingestion of contaminated plants.

Instream sediments. Instream sediments are intermediate in mobility. They may remain in one place for long periods of time, but may migrate downstream when the rate of surface water flow is high enough, as in flood events (TVA 1985). Because of their relative stability, instream sediments possess a moderately long-term accumulation of released particulates and contaminants that sorb to particulates. They may cause exposure by both ingestion and dermal contact for crayfish, bottom-feeding fish, and benthic macroinvertebrates. Suspended sediments may also expose biota living predominantly in the upper water column; instream sediments may be a minor exposure medium for piscivorous mammals and birds through direct contact and ingestion. Stream sediments deposited by floods on plants may be an important route for exposure of plants by foliar absorption and for herbivorous biota by ingestion of sediments on plant surfaces.

Shallow groundwater. Groundwater flow in EFPC is described by a stormflow zone flow model in which intermittently saturated zones on the ridge slopes drain downslope, forcing groundwater to flow to discharge points or to the aquifer at the valley floor (Moore 1988; LWA

1991). In periods of high creek flow, groundwater may be recharged from the surface water, allowing soluble contaminants to reenter the aquifer.

Shallow groundwater may provide transient exposure of dissolved contaminants to plant roots, burrowing animals, insects, or worms. Groundwater at seeps can also be a source of exposure for other animals. However, groundwater is primarily a transport medium for contaminants entering the soil from surface runoff, for leachates from soil, and for dissolved contaminants entering surface water where discharge occurs. Historical (Carmichael 1989) and Phase Ia sampling have revealed the presence of low levels of contamination of shallow groundwater by inorganic and organic contaminants as well as by low levels of uranium. However, most of the contaminant appears to have been associated with suspended particles rather than soluble species.

Deep groundwater. Because of geological barriers between shallow groundwater and the deeper aquifers, deep groundwater is assumed to be uncontaminated (LWA 1991). Further, because it is relatively inaccessible, deep groundwater is not likely to be a source of contaminants to the biota being assessed.

Soil. Soil is a primary source of contaminants to terrestrial biota. Relatively immobile, it can accumulate contaminants deposited by surface runoff from point and nonpoint sources. These potentially include commercial sources such as fuel transfer businesses and automobile dealerships, as well as streets and parking lots, golf courses, home lawns and gardens, and the Oak Ridge Sewage Treatment Plant. Contaminants may also be deposited by wet and dry deposition from offsite sources (e.g., radioactive fallout). During storms, soil may erode into EFPC, where it can be transported downstream, subsequently settling out as instream sediment or as new alluvial deposits or exiting the system into Poplar Creek. Soluble contaminants may leach from the soil into surface runoff or shallow groundwater, which can transport them to the creek.

In contrast to surface water, which responds quickly to changes in concentrations of contaminants released from the Y-12 Plant, the EFPC floodplain soils are relatively stable. Thus, they may accumulate relatively insoluble contaminants over a long period of time. Animals that live on or above the soil surface would be exposed only to contaminants in exposed surface soil. Plants and burrowing animals would also be exposed to surface and subsurface contaminants. Small mammals, terrestrial insects, and earthworms are among the exposed indicator organisms.

Air. Contaminants may be transported by air if they are volatile or if they are suspended as particulate material. They can be inhaled by animals or absorbed directly by animals and by

plants through surface deposition or transpiration. During Phase Ia sampling, volatile organics were not found in surface water or soil in concentrations high enough to justify their inclusion as contaminants of concern (Sect. 5.1). Volatile mercury has been observed in new boreholes during sampling, but the observed ambient mercury levels in air have not indicated that inhaled air is a significant exposure source. In addition, the prevailing winds, which tend to parallel the ridges and valleys, would contain within Bear Creek Valley most volatile and airborne particulate contaminants from the Y-12 Plant rather than transport them across Pine Ridge and East Fork Ridge to the floodplain. Transport of contaminants from EFPC floodplain soils as airborne particulates is expected to be of minor importance because of the extensive ground cover and relatively moist conditions in the floodplain.

Endpoint biota (indicator organisms). The endpoint biota or indicator organisms reflect exposures to contaminants from the Y-12 Plant, the EFPC floodplain, or offsite sources by different exposure routes (indicator organisms are discussed in Sect. 6.1.4.3). The relationship of each indicator to a source may be direct (i.e., where the source is the medium of exposure), or indirect (i.e., where the source is not in direct contact with indicator organisms, but rather the exposure occurs through a transport medium). For example, fish may be directly exposed to contaminants in sediment by ingestion of sediment while feeding or by direct exposure of eggs deposited on the creek bottom. Periphyton may be exposed indirectly to contaminants in sediments if the sediments are disturbed and suspended in the water column or if the contaminants are leached into the water. In contrast, there is no significant direct or indirect exposure of earthworms to sediment contaminants—this pathway is incomplete or negligible. The relationship of indicators to sources is summarized in Table 6.11.

6.2.1.2 Mechanisms of chemical transformations

Because the toxicity of contaminants may depend strongly on their chemical form, it is important to understand what mechanisms of chemical transformation may operate in the study area. The most important contaminant of concern is mercury, which was originally used at Y-12 in the relatively nontoxic elemental form. Acid treatment and other oxidation processes may have converted some of the elemental mercury to mercury salts. Bacterial metabolism, especially in organisms that produce excess amounts of methylcobalamin, results in the formation of methylmercury, which is neurotoxic. Methylmercury formation appears to be more pronounced in the bottom sediments of Lake Reality than in other parts of the EFPC system. Methylmercury formed in Lake Reality appears to be incorporated into protists, passed through the food chain to fish, and presumably ends up in piscivorous mammals and birds.

Table 6.11. Dominant mode of exposure of indicator organisms to contaminated source media in EFPC

Indicator biota	Medium			
	Surface water	Instream sediment	Groundwater	Soil
Fish	D	D/I	*	*
Benthic invertebrates	D	D	*	*
Aquatic insects	D	D	*	*
Crayfish	D	D	*	*
Periphyton	D	I	*	*
Small mammals	D	*	I	D
Birds	D	I	I	I
Terrestrial insects	*	*	I	D
Earthworms	*	*	I	D
Plants	D	D	I	D

D = Direct exposure.
* = Incomplete pathway or considered negligible.
I = Indirect exposure.

Surface water. Transformations of mercury are probably the most significant chemical transformations occurring in surface water. Microbial populations in the waters of Lake Reality and EFPC include microbes with the ability to demethylate methylmercury and to reduce mercury salts to elemental mercury (TVA 1985). Elemental mercury at low concentrations will readily volatilize from the stream in the well-aerated riffle areas. Therefore, the concentrations of all forms of soluble mercury in surface water are low under base flow conditions. Oxidation/reduction reactions of other inorganics are possible, but because the concentrations of other analytes are low, they are probably of less importance than chemical transformations of mercury.

Organic contaminants are also transformed by chemical and biological processes. Complex organics such as pesticides may be hydrolyzed, oxidized in riffles, or degraded by photolytic reaction in sunny areas. Some organic contaminants may be metabolized by microbes in the surface water, although to date no evidence has been presented of microbial degradation of organics in EFPC. Some organics may be degraded by higher organisms. For example, fish degrade PAHs to simpler compounds, and it is likely that other contaminants are detoxified by biota.

The distribution of contaminants in EFPC surface water may change even if the compounds themselves are not altered. PCBs are extremely stable compounds and are slow to degrade in the environment (EPA 1983). In the water column, hydrophobic PCBs preferentially partition into lipids and other organic apolar compounds or are physically adsorbed on particulate organic matter. Transfer of PCBs on microparticulate materials and into phytoplankton is well documented, as is partitioning from aqueous solutions into algal lipids. PCBs accumulate to a higher extent in oily fish such as carp than in fish with lower fat content such as sunfish (Loar 1992). Piscivorous birds and animals also accumulate PCBs and other organics; accumulation of mercury and PCBs in heron eggs on the Clinch River has been documented (MacIntosh et al. 1992).

Instream sediments. In the fish corral study described in Sect. 6.3.2.3 and in Appendix Q, an attempt was made to determine whether contaminated sediment or contaminated water is the major source of exposure for sunfish in EFPC. Fish were exposed to (1) upper EFPC water alone, (2) upper EFPC water with stream sediment, (3) uncontaminated groundwater alone, and (4) uncontaminated groundwater with stream sediment. The study showed that mercury from the water of upper EFPC became associated with sediments, resulting in the production of methylmercury. When sediments were present, the average ratio of methylmercury to total mercury in sediment was $\sim 6 \times 10^{-4}$. The ratios of methylmercury to total mercury in

biota were $\sim 1 \times 10^{-3}$ in algae, periphyton, and macrophytic plants; 0.8-1 in insects and snails; and ~0.8 in sunfish muscle. These results suggest that methylation of mercury by sediment-dwelling microbes and bioconcentration of methylmercury by invertebrates in the food chain may be a major mechanism for exposing fish to methylmercury.

Instream sediments are also a likely location for biodegradation of organic contaminants by microbial metabolism. The studies reported here were not designed to measure these activities but rather to measure baseline ecological effects because of the chemical processes.

Shallow groundwater. Biodegradation of dissolved contaminants by microbial processes may occur in shallow groundwater, both in aerated zones and in anaerobic microhabitats. Analysis of shallow groundwater has revealed the presence of acetone in some samples. Acetone may be a degradation product of other organic contaminants.

Deep groundwater. The predominant chemical transformations in deep groundwater will most likely be oxidation/reduction reactions of metal ions. If halogenated organic contaminants are present in deep groundwater, they may be subject to microbial metabolism, including dehalogenation and biochemical oxidation.

Soil. Chemical and biochemical reactions of contaminants in soil are complex. Soil particles are a key site for transient or long-term removal of contaminants from shallow groundwater or surface runoff. Contaminants that are poorly soluble in water are likely to sorb strongly to soil particles. Soil particles are also the site of most soil microbial activity. Therefore, organic contaminants may be concentrated from runoff water or shallow groundwater onto particles where they are susceptible to microbial degradation. Products of degradation reactions may be more soluble, allowing them to leach into groundwater. Inorganic contaminants may react with mineral or organic components of the soil, changing in oxidation or becoming immobilized as precipitates. The majority of the mercury in EFPC floodplain soils seems to be in a precipitated or poorly soluble complex form. Floodplain soils do not seem to be an important component in the formation of methylmercury (Revis et al. 1989a).

Air. Volatile organic contaminants may be subject to photolytic and hydrolytic degradation in the air. In addition, mercury and other volatile compounds may be sorbed to airborne particulate matter. However, because ambient temperatures are not high enough to volatilize organic contaminants, the concentrations of contaminants of concern in air are assumed to be small. Their degradation products are, therefore, not likely to contribute significant risk to the ecosystem.

6.2.1.3 Routes of contaminant migration and transport

Y-12 Plant. Contaminants may be transported from the Y-12 Plant by spills, erosion, or surface runoff into upper EFPC (Fig. 6.16). This water passes through Lake Reality and exits the Y-12 Plant site, entering the study area. Contaminants may be dissolved in the creek water or transported in association with organic and inorganic particulate matter. They also may be incorporated in organisms. Transport of contaminants from the Y-12 Plant to the EFPC floodplain system by air or by groundwater is not considered to be significant (LWA 1991a).

Historical evidence (TVA 1985) as well as the results of Phase Ia sampling and analysis have shown that most of the contaminants of concern leaving the Y-12 Plant are transported in association with particulate materials. Early studies (TVA 1985) suggested that mercury transported in surface water was preferentially associated with small particulate material. However, size distribution studies of EFPC floodplain soils do not show any size dependence of mercury concentration. Because the amount of particulate material suspended in surface water increases dramatically during storms (TVA 1985), the rate of contaminant transport varies with the storm hydrograph. After an initial brief increase, contaminant concentration rapidly falls as the washed-out contaminants are diluted by surface runoff.

EFPC floodplain soils. Contaminants may be transported within the EFPC floodplain by wind transport of particulates or by emanation of volatile compounds. Because of the extensive ground cover on most of the floodplain, wind is a minor mechanism of contaminant transport. Volatile contaminants are not widely distributed in surface soils, although mercury vapor and organics may be found in soil gas and, thus, may diffuse into the air.

Erosion and surface runoff are the major pathways for transport of contaminants within the EFPC floodplain and out of the floodplain system. Nonpoint sources throughout the watershed contribute contaminants that may accumulate as sediment in the tributaries and be washed into EFPC. Instream sediments are scoured and redeposited further downstream or are washed into Poplar Creek and the Clinch River. Similarly, soil contaminants are transported by bank erosion, which deposits contaminants as sediment into EFPC, especially during storms when water flow is high. A correlation has been demonstrated between both flow rates and sediment concentration and bulk transport of mercury in EFPC (TVA 1985).

Shallow groundwater is thought to move laterally from the ridges to the EFPC floodplain and could transport contaminants leached from the soil. Because of the large areas of watershed and the apparent importance of subsurface movement of water, shallow groundwater is potentially an important means of transport of leachate into floodplain soils, from floodplain soil to surface

water, and from surface water to floodplain soil. However, analysis of shallow groundwater has shown that the concentrations of dissolved contaminants are low, suggesting that this is a minor pathway for contaminant transport. The deep aquifer also does not appear to contain dissolved contaminants at significant concentrations; therefore, it is not an important pathway for contaminant transport.

Uptake of contaminants by plants and animals may result in migration of contaminants from place to place within the floodplain and out of the system. For example, soil contaminants may be incorporated by worms and insects, which are consumed by small mammals and birds, which may move about in the floodplain and outside it. Plants may incorporate contaminants by root absorption and subsequently be eaten or die and be washed downstream to be redeposited or removed from the system. The movement of biota, especially animals, is probably an important mechanism for transport of contaminants in the EFPC floodplain.

6.2.1.4 Areas of exposure and habitats

To facilitate evaluation of exposures in different EFPC habitats, the study area was divided into sections. The divisions were based on the EFPC sampling grid; 17 sections were created, each comprising one km from west to east on the sampling map (Fig. 6.17). Section 1 begins just below the confluence of EFPC with Poplar Creek, and Section 17 includes areas of exposure below Lake Reality. These sections are different from the sections used in Chapters 3 and 5. The discussion below summarizes the distribution of the following areas of exposure in the EFPC ecosystem: air, groundwater, surface water and sediments (aquatic habitats), soil and vegetation (terrestrial habitats), and wetlands.

Air. There is insufficient information available to calculate the size of any discrete units of exposure to airborne contaminants. It is assumed that airborne contaminants have had and will continue to have little impact on the EFPC ecosystem.

Groundwater. Groundwater is not considered a significant source for exposure to contaminants. Therefore, it is not necessary to identify discrete areas of potential exposure to groundwater.

Surface water and sediments (aquatic habitats). Surface water habitats were identified and classified by type for the entire length of EFPC. Four types of aquatic habitat were identified [a total of ~26 ha (65 acres)], and these are defined below:

other: log jams and other debris, small impoundments.

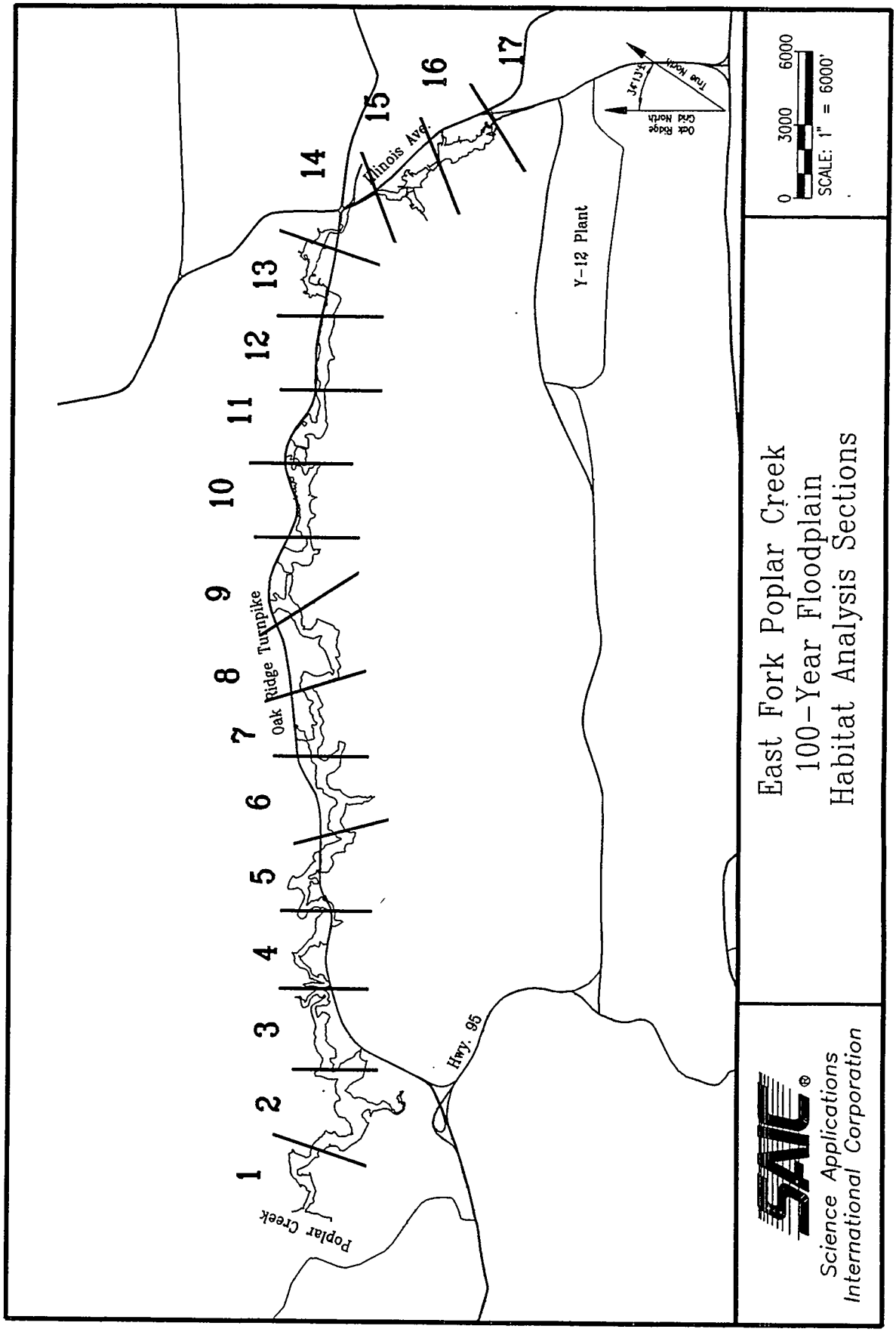


Fig. 6.17. Map of 17 sections for habitat analysis.

pool: stream section of deeper, slower-moving water which occurs in areas of relatively flat gradients. This usually occurs at bends in a channel. Velocity of current is reduced and can exhibit back-current at surface. Pools generally have well-defined boundaries with bowl-shaped bottom areas. They are characterized by deposition; silt and other suspended materials tend to settle, forming a soft substrate suitable for burrowing worms and nekton.

riffle: section consisting of faster, shallower water than found in pool areas and occurring mostly in higher gradient reaches. Velocity of current is great enough to keep the bottom clear of silt and other loose materials, providing a firm substrate which is occupied largely by specialized benthic macroinvertebrates, periphytic organisms, and some fish species. Distinguished from run areas by turbulent flow with disturbed/broken surface tension.

run: section similar to riffle areas, but usually lower in velocity and gradient than riffle areas, and exhibits a more laminar flow of water with no or very little disruption of surface tension. Runs generally have a more constant depth than pools.

Table 6.12 displays the areas of distinct aquatic habitat units (i.e., other, pools, riffles, runs). Table 6.12 also shows that the total area of runs is much greater than that of riffles, pools, or other habitats. There are many riffles and pools, but their area is small compared to the area for runs. The distribution of aquatic habitats in each of 17 1-km sections is shown in Fig. 6.18. The area of runs in each section increases downstream as the stream widens, and the majority of the pool areas are in the downstream half of the creek. There are riffle areas starting in the uppermost sections and continuing downstream to the beginning of the creek.

Concentrations of contaminants in sediments were determined by sampling instream sediment at 200-m (660 ft) intervals and combining three successive samples. Therefore, each sample represents a stream reach of ~600 m (1980 ft). Results of this analysis are given in Sect. 3.2.2.2. Concentrations of mercury in the instream sediments during this sampling period are shown in Fig. 6.19.

Soil and vegetation (terrestrial habitats). Exposure of biota to contaminants in soil and attendant vegetation varies according to the feeding habits of the organisms. Terrestrial habitats in the EFPC floodplain were identified and their areas were tabulated to make it easier to estimate exposure of biota of different types to contaminants in soil (Fig. 6.20). Terrestrial habitats were

Table 6.12. Aquatic habitat areas (AC) by segment

Habitat areas (AC)					
Segment	**Other**	**Pool**	**Riffle**	**Run**	**All**
All	0.41	7.82	8.46	48.36	65.05
17	0.00	0.03	0.17	0.60	0.80
16	0.06	0.05	0.60	1.08	1.79
15	0.01	0.01	0.33	1.59	1.93
14	0.00	0.05	0.09	2.02	2.15
13	0.00	0.00	0.04	2.47	2.52
12	0.01	0.06	0.20	2.22	2.50
11	0.01	0.29	0.66	1.80	2.75
10	0.01	0.28	0.80	1.91	3.01
9	0.01	0.72	0.38	3.19	4.29
8	0.02	0.60	0.58	3.78	4.98
7	0.10	1.23	0.30	1.46	3.09
6	0.01	0.78	0.27	3.62	4.68
5	0.13	1.18	1.18	4.69	7.18
4	0.01	1.11	1.21	3.06	5.38
3	0.03	0.56	0.57	4.05	5.21
2	0.00	0.19	1.05	5.60	6.84
1	0.01	0.69	0.00	5.21	5.91
0	0.00	0.00	0.03	0.01	0.05

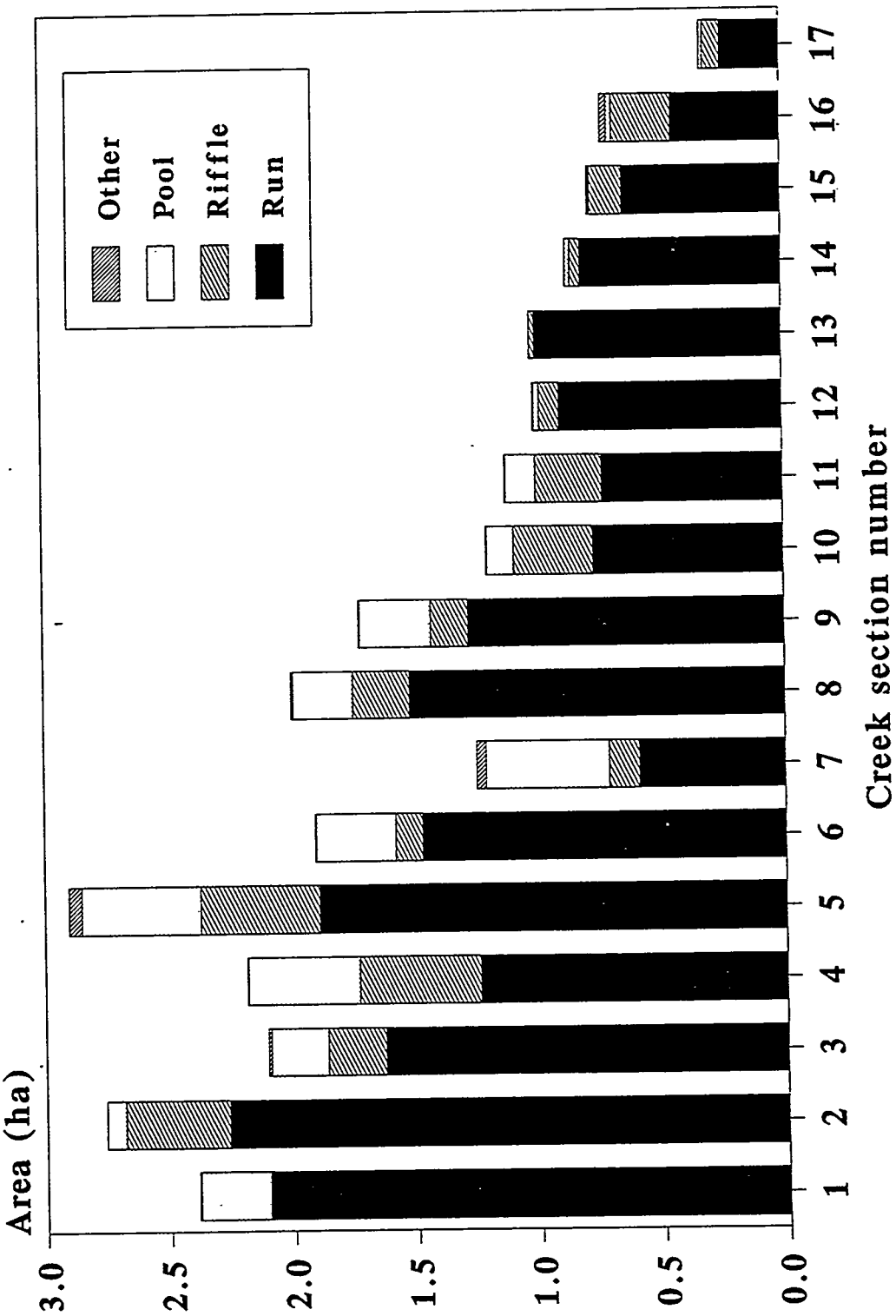


Fig. 6.18. Aquatic habitat areas by stream section.

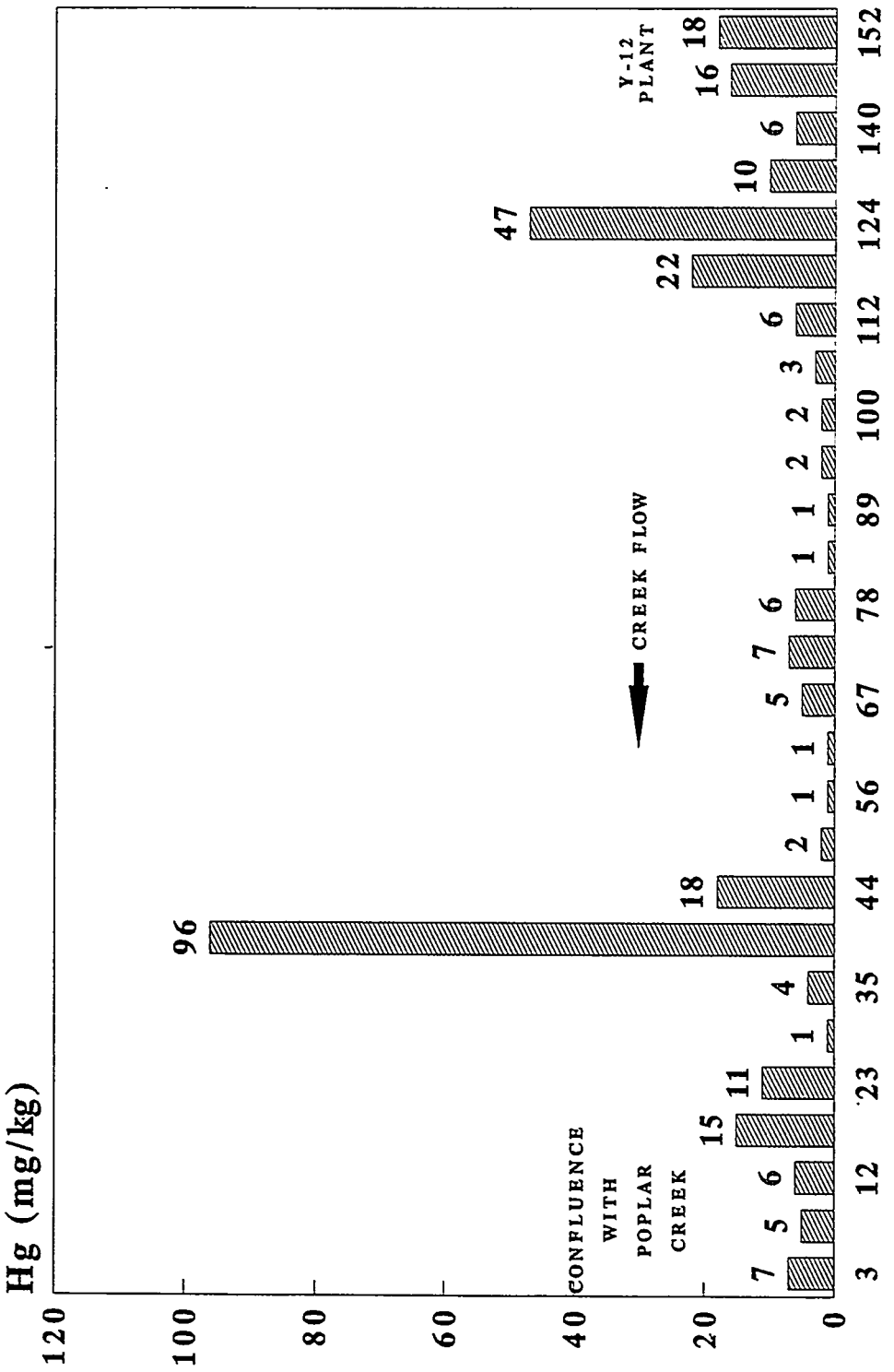


Fig. 6.19. Mercury in EFPC sediment.

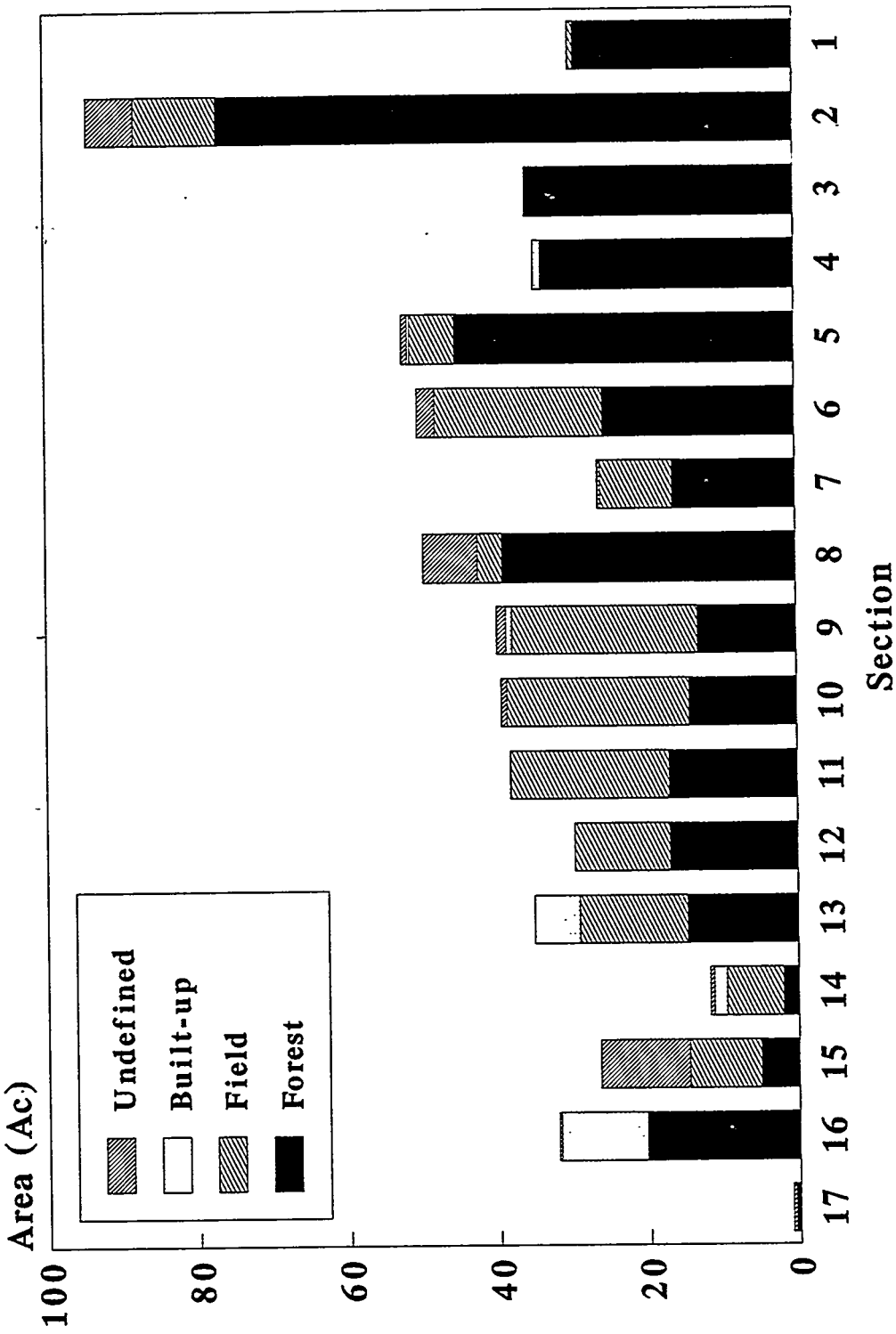


Fig. 6.20. Terrestrial habitats in the EFPC hundred-year floodplain by stream section.

identified and classified by type for the entire length of EFPC (using the 100-year floodplain as the boundary. A total of 255.30 ha (628.05 acres) of terrestrial habitat were identified. Table 6.13 displays the areas of the following distinct habitat units on a section-by-section basis:

- **Bottomland Hardwood Forest:** Forest dominated by tree species well-adapted to growth in deep, alluvial soil along a stream subject to periodic flooding.
- **Commercial:** Pavement, parking lot, or rip-rap.
- **Fill:** Area of natural floodplain that has been filled but is not currently in commercial use.
- **Grass:** Grass that is cut periodically. It is not necessarily a monoculture of grass; it may have many forbs present also. It is not used for recreation.
- **Hardwood Plantation:** Planted forest of nonconiferous trees. Understory varies depending on the degree of canopy closure.
- **Lawn:** Maintained lawn in recreational use (e.g., a golf course or residential lawn).
- **Old Field:** Former agricultural or pasture lands dominated by grasses and forbs. Tree and shrub seedlings typically are present. It is neither grazed nor mowed.
- **Pasture:** Open, grassy areas used for grazing by cattle or horses.
- **Pine Plantation:** Planted forest of pine trees, typically loblolly and white pine. Understory is typically sparse in mature stands, but more diverse if some areas have been harvested without replanting.
- **Right-of-way (ROW):** Utility ROW, typically mowed once a season, sometimes mowed every other year. Grasses, forbs, shrubs, and small trees are present.
- **Transportation:** Road.
- **Upland Forest:** Mixed forest growing on the edge of the floodplain or in upland areas adjoining the floodplain.
- **Undefined:** Habitat not defined because of absence of aerial photography or other such reason.

Table 6.13. Terrestrial habitat areas (AC) by segment

Habitats Areas (AC)														
Segment	Bottom Land	Commercial	Fill	Grass	Hardwood plantation	Lawn	Old field	Pasture	Pine	Right of way	Transportation	Upland	Undefined	All
ALL	326.99	19.05	1.43	38.62	4.42	4.37	61.48	62.03	15.79	0.98	1.32	59.74	31.81	628.02
17	0.61	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.34	0.95
16	20.29	10.91	0.64	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.30	32.14
15	5.01	0.01	0.00	7.55	0.00	0.00	2.01	0.00	0.00	0.00	0.00	0.00	11.97	26.55
14	2.06	1.63	0.00	4.29	0.00	0.00	3.20	0.00	0.00	0.00	0.00	0.00	0.56	11.74
13	14.75	6.05	0.00	9.52	0.00	0.00	4.80	0.02	0.00	0.00	0.00	0.00	0.00	35.14
12	17.02	0.00	0.00	2.07	0.00	0.00	10.64	0.00	0.00	0.00	0.00	0.00	0.00	29.73
11	17.09	0.07	0.00	1.37	0.00	0.00	10.72	9.05	0.00	0.00	0.00	0.00	0.01	38.31
10	14.39	0.00	0.00	0.00	0.00	0.00	2.70	21.60	0.00	0.00	0.00	0.00	0.76	39.45
9	12.45	0.03	0.79	0.04	0.00	0.00	6.59	18.23	0.00	0.00	0.00	0.64	1.21	39.98
8	33.89	0.00	0.00	0.96	0.00	0.00	2.36	0.00	0.00	0.00	0.00	5.34	7.26	49.81
7	16.39	0.35	0.00	8.10	0.00	0.28	1.00	0.28	0.00	0.00	0.00	0.00	0.02	26.42
6	25.70	0.00	0.00	4.72	0.00	4.09	2.98	10.63	0.00	0.00	0.00	0.01	2.32	50.45
5	32.96	0.00	0.00	0.00	0.00	0.00	2.77	2.22	1.42	0.98	0.32	11.05	0.77	52.49
4	18.62	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.99	15.18	0.00	34.79
3	20.90	0.00	0.00	0.00	0.00	0.00	0.00	0.00	3.38	0.00	0.00	11.61	0.00	35.89
2	51.01	0.00	0.00	0.00	4.21	0.00	11.03	0.00	9.61	0.00	0.00	12.10	6.29	94.25
1	23.85	0.00	0.00	0.00	0.21	0.00	0.68	0.00	1.38	0.00	0.00	3.82	0.00	29.94

These habitats can be grouped into three major types: (1) forests, consisting of all types of forests, hardwood, bottomland, upland and pine; (2) fields consisting of old field, ROW, pasture, grass and lawn; and (3) built-up, consisting of commercial, transportation, and fills.

The majority of the terrestrial habitats are bottomland hardwoods (> 50%), with ROW and transportation corridors (less than 0.4%) being the smallest two types. The other habitats are intermediate in areas as shown in Fig. 6-20.

EFPC Wetlands. Wetlands are those areas inundated or saturated by surface or ground water at a frequency and duration sufficient to support, and that under normal circumstances do support, a prevalence of vegetation typically adapted for life in saturated soil conditions (40 *CFR* 230.3; 33 *CFR* 328.3). Wetlands generally include swamps, marshes, bogs, and similar areas. Wetlands possess three basic attributes - wetland hydrology, hydrophytic vegetation, and hydric soils. All three criteria must be present for an area to be defined as a wetland.

The U.S. Army Corps of Engineers (COE) identified 17 wetlands in and around the floodplain of EFPC (COE 1992). These wetlands ranged in size from 0.004 ha (0.01 acres) to 1.14 ha (2.81 acres) (Table 6.14). Of the 17 wetlands inventoried, only 16 are actually located within the 500-year floodplain of EFPC. Of a total of 4.79 ha (11.83 acres) of wetlands inventoried, 4.78 ha (11.82 acres) are actually in the 100-year floodplain of EFPC.

These wetlands occur in three habitat types within the floodplain: bottomland hardwood, scrub-shrub, and permanently ponded. Approximately 2.7 ha (6.67 acres) of wetlands are found in the bottomland hardwood forests comprised principally of green ash (*Fraxinus pennsylvanica*), American sycamore (*Platanus occidentalis*), and boxelder (*Acer negundo*); 1.91 ha (4.71 acres) are dominated by shrub such as dogwood (*Cornus* spp.), alder (*Alnus* spp.), or buttonbush (*Cephalanthus* spp.); and 0.18 ha (0.44 acre) is a shallow pond dominated by duckweed (*Lemna* spp.). The wetlands in the EFPC floodplain provide highly productive wildlife habitat. Other wetland functions include floodwater attenuation and sediment (and contaminant) retention. The following narrative provides descriptions of each wetland in the EFPC floodplain from Y-12 to the confluence of EFPC and Poplar Creek.

Wetland 1 - 0.158 ha (0.39 acres). Wetland 1 is located on the east bank of EFPC near Transect 152, coordinate N 9800 m (32144 ft) adjacent to the car wash wetland on Scarboro Road in Pine Ridge Gap (Fig. 6.37). Transect 152 traverses the south end of the wetland. Mercury values range from 1170, 332, and 160 ppm 0 to 0.4 m, 0.4 to 0.8 m, and 0.8 to 1.2 m (0-16, 16-32, and 32-48 in., respectively) at Station 00 to 0, 0, and 0 ppm at Station 02. A CLP sample at Station 00 [0 to 0.4 m (0-16 in.)] had mercury concentrations of 1300 ppm at Station 00 and

Table 6.14. Potential jurisdictional wetlands in the East Fork Poplar Creek floodplain

Wetland ID	Wetland location[a]	Land use	Contamination level[b]	Wetland area (acres)
1	Creek mile 14.1 right bank	DOE	C	0.39
2[c]	Creek mile 14.0 left bank	Open	NC	0.01
3	N 33468 left bank	Open	C	0.57
4	N 33806 left bank Station W-12	Open	NC	0.56
5	N 34452 right bank Station E-06	Open	C	0.85
6	N 58069 right bank Station N-16	Residential	NC	0.09
7	E 54788 right bank Station N-08	Open	C	2.12
8	E 53804/E 53476 left bank	Open	C	0.91
9	E 43306 right bank	Open	NC	2.81
10	E 42324 left bank	Open	C	0.12
11	E 40024 right bank	Open	NC	0.21
12	E 30674 left bank	DOE	NC	0.45
13	E 20339 right bank	DOE	C	0.61
14	E 20339 right bank, Station N-14	DOE	C	0.86
15	E 20011 left bank	DOE	NC	0.08
16	E 19682 left bank	DOE	NC	0.39
17	E 19355 right bank	DOE	NC	0.80
TOTAL				11.83

[a]Wetland locations are identified according to creek mile or Phase Ib soil sampling transects; right or left bank refers to direction when facing downstream. Station numbers refer to soil sampling locations along Phase Ib soil sampling transects.
[b]C = Contaminated ($\geq$ 50 μg/g Hg); NC = Not Contaminated ($<$ 50 μg/g Hg).
[c]Wetland 2 is not in the 500-year floodplain of EFPC.

141 ppm at Station 02 [0 to 0.4 m (0-16 in)]. Triangulation shows the entire wetland to be contaminated. Transect 151 would have crossed the north end of the wetland, but was never sampled because no access agreement was ever obtained. Vegetation in the wetland is a mixture of bottomland hardwoods in the southern end and shrubs in the wetter northern end. The northern end typically has standing water all year long. The wetland appears to have formed as the result of seasonal floods and poor surface drainage that has apparently been modified by filling the floodplain to allow construction of the car wash. Wetland 1 is on DOE property.

Wetland 2 - 0.004 ha (0.01 acres). Wetland 2 is located along Transect 150, coordinate E 1000 m (32800 ft) outside the 500-year floodplain in what appears to be an old borrow pit. Wetland 2 is about 200 m (660 ft) north (downstream) of Wetland 1 on the west bank of EFPC. Vegetation consists of emergent marsh vegetation (cattails, sedges, rushes). No soil samples were taken because the wetland is so far outside the floodplain. The wetland appears to have formed in a small seep or spring in the borrow pit that may have been enhanced by modification of the topography by recent earth moving. Wetland 2 is on privately owned land and is classified in the open land use category.

Wetland 3 - 0.23 ha (0.57 acres). Wetland 3 is located on the west side of EFPC on Transect 149, coordinate N 10100 m (33128 ft) and Transect 148, coordinate N 10200 m (33456 ft) behind NOAA's Atmospheric Diffusion Laboratory (NO Site) on South Illinois Avenue (Fig. 6.36). Transect 149 traverses the south end of the wetland, but no samples were taken in the wetland. Transect 148 cuts through the northern lobe of the wetland, and one sample was taken in the wetland. No results are available from the 0 to 0.4 m (0-16 in.) sample because data were rejected by data validators per Level IV criteria, but the data are expected to meet Level II criteria and, therefore, be used. The reason for the rejection is not known. Mercury values were 0 ppm for two deeper samples in the same hole [0.4 to 0.8 m and 0.8 to 1.2 m (16-32 in. and 32-48 in.)]. Despite the rejected data in the wetland, triangulation data indicate the wetland is contaminated. Vegetation is a mixture of bottomland hardwoods. The wetland appears to have formed as a result of a combination of seasonal floods, small seeps or springs, and poor surface drainage. Wetland 3 is on privately owned land and is classified in the open land use category.

Wetland 4 - 0.23 ha (0.56 acres). Wetland 4 is located on the west side of EFPC at the NO site along Transect 147, coordinate N 10300 m (33784 ft) near the USGS monitoring well transect (Wells AN:D-1 through AN:D-6) (Fig. 6.36). All analytical results were rejected by validators on the west side of the creek for Level IV criteria, but the data are expected to meet Level II criteria. Despite the rejected data in the wetland, triangulation data indicate the wetland is not contaminated. Vegetation is a mixture of bottomland hardwoods. This wetland appears to have

formed as a result of a combination of seasonal floods, small seeps or springs, and poor surface drainage. Wetland 4 is on privately owned land and is classified in the open land use category.

Wetland 5 - 0.34 ha (0.85 acres). Wetland 5 is located near Transect 145, coordinate N 10500 m (34440 ft) near the NO site (Fig. 6.36). Wetland 5 is in the floodplain on the east side of EFPC. Transect 145 traverses the southern boundary of the wetland and samples were taken from the top 0.4 m (16 in.) of soil. Mercury values ranged from 124, 287, 41, to 0 ppm from west to east. Vegetation is bottomland hardwoods (mostly young second growth green ash). Well AN:E-4 is located near the western end of the wetland. Triangulation data indicate the wetland is contaminated with mercury. This wetland is also threatened by recent commercial development in the area. Seasonal flooding and poor surface drainage are the primary factors influencing the hydrology of this wetland. Wetland 5 is on privately owned land and is classified in the open land use category.

Wetland 6 - 0.04 ha (0.09 acres). Wetland 6 is located on Transect 122, coordinate E 17700 m (58069 ft) north of EFPC behind Robertsville Jr. High and the Robertsville Baptist Church (Fig. 6.33). Transect 122 traverses the western end of the wetland. All mercury data were rejected by validators for Level IV criteria, but the data are expected to meet Level II criteria. Because the wetland is on the outer edge of the EFPC floodplain, the creek has probably never flooded the wetland and it is probably uncontaminated. Vegetation is dominated by marsh plants and shrubs adjacent to a nonwetland dominated by bottomland hardwoods. The hydrology of the wetland appears to be controlled by drainage from a seep or spring located behind the church. Wetland 6 is on privately owned land and is classified in the residential land use category.

Wetland 7 - 0.86 ha (2.12 acres). Wetland 7 is located along Transect 111, coordinate E 16600 (54448 ft) at the BR site across the Oak Ridge Turnpike from the Bruner and Four Oaks Shopping Centers (Fig. 6.32). The wetland is bounded on the north by Oak Ridge Turnpike and on the south by EFPC. The transect nearly bisects the wetland into western and eastern halves. Mercury concentrations range from 6 ppm at Station 00, 31 ppm at Station 02, 259 ppm at Station 04, 221 ppm at Station 06, 49 ppm at Station 08, to 0 ppm at Station 10. The wetland begins at about Station 02. Vegetation is a young bottomland hardwoods stand (mostly green ash). The wetland appears to have formed as a result of seasonal flooding and poor drainage. The surface is often inundated for several days after heavy rains and floods. It appears an attempt was made to drain this wetland in the past; a small drainage ditch leading to EFPC is located near the western end of the wetland. Construction of Oak Ridge Turnpike may have affected the northern boundary, but it is hard to tell. This wetland is very similar to Wetland 5. Wetland 7 is on privately owned land and is classified in the open land use category.

Wetland 8 - 0.37 ha (0.91 acres). Wetland 8 is located on Transect 109, coordinate E 16400 m (53792 ft), and Transect 108, coordinate 16300 m (53464 ft), at the BR site (Fig. 6.32). Wetland 8 is located on the south side of EFPC near the second USGS monitoring well transect (Wells AN:D-X through AN:D-XX). The wetland is bounded on the south by East Fork Ridge and on the north by EFPC. The two soil transects roughly divide the wetland into thirds. Mercury concentrations in the wetland on Transect 109 are 102 and 238 ppm [0 to 0.4 m (0-16 in.)], respectively, at Stations 04 and 06. The wetland begins between Stations 02 and 04 and ends around Station 06 on Transect 109. The wetland begins at Station 02 and ends near Station 08 on Transect 108. Mercury values on Transect 108 are 559, 13, and 11 ppm at Station 02 [0 to 0.4 m, 0.4 to 0.8 m, and 0.8 to 1.2 m (0-16, 16-32, and 32-48 in.)]; 51 ppm at Station 04; 1070, 24, and 0 ppm at Station 06; and 107, 0, and 0 ppm at Station 08. Triangulation data show the entire wetland is contaminated. Vegetation is similar to that at Wetland 7 (bottomland hardwoods) but slightly younger with a little more diversity of species. The wetland appears to have formed as a result of seasonal flooding and poor drainage. The surface is often inundated for several days after heavy rains. Wetland 8 is on privately owned land and is classified in the open land use category.

Wetland 9 - 1.14 ha (2.81 acres). Wetland 9 is located north of EFPC on Transect 77, coordinate E 13200 m (43296 ft), which cuts the wetland roughly in half (Fig. 6.29). No sampling was done in the wetland. Apparently the transect passed critical elevation (above 100-year flood elevation) very close to the creek bank, but then topography quickly drops back down into the wetland. Drainage from the wetland is constricted by the topography, which may possibly have prevented contaminants carried by EFPC water backing up into the wetland during floods. The wetland is bounded on the north by Oak Ridge Turnpike, on the east by Big Turtle Park, and on the west by the Oak Ridge Sportsmen's Association (ORSA) property. Vegetation consists mostly of shrubs and grasslike herbaceous plants with a few small trees scattered throughout the area. The wetland has apparently formed as a result of flow from perennial seeps or springs restricted by local topography. The wetland may also receive additional water from two small tributaries and, possibly, some backwater from EFPC during floods. Wetland 9 is on privately owned land and is classified in the open land use category.

Wetland 10 - 0.05 ha (0.12 acres). Wetland 10 is located on Transect 74, coordinate E 12900 m (42312 ft), on ORSA property (Fig. 6.28). The wetland is roughly crescent-shaped parallel to the outside edge of a meander bend in EFPC. Transect 74 cuts across the middle of the wetland. The wetland begins close to Station 00 on the south bank of EFPC and ends between stations 02 and 04. The mercury value at Station 00 was 23 ppm with 0 ppm at each remaining

station on the transect. Vegetation is bottomland hardwoods. The wetland apparently formed in an old meander bend and receives water from flood events; surface drainage is poor. This area may not actually be a wetland. Access to the site was limited so the broadest assumptions on the presence of the three wetland criteria were used to make the wetland determination. Wetland 10 is on privately owned land and is classified in the open land use category.

Wetland 11 - 0.09 ha (0.21 acres). Wetland 11 is located along Transect 67, coordinate E 12200 m (40016 ft), on ORSA property near Gum Hollow Road (Figs. 6.28 and 6.27). Transect 67 roughly bisects the wetland. All mercury data were rejected by validators for Level IV criteria, but the data are expected to meet Level II criteria. However, triangulation data indicate the wetland is not contaminated. The wetland is located north of EFPC near the point where two intermittent tributaries (TQ and TG) enter EFPC. Vegetation is bottomland hardwoods. This area may not actually be a wetland. The broadest assumptions on the presence of the three wetland criteria were used to make the wetland determination because access to the site was limited. Wetland 11 is on privately owned land and is classified in the open land use category.

Wetland 12 - 0.18 ha (0.45 acres). Wetland 12 is located on Transects 38 and 39, coordinates E 9300 m and E 9400 m (30504 ft and 30832 ft) (Fig. 6.25). The two transects split the wetland roughly into thirds. Wetland 12 is located on the south side of EFPC and Highway 95/58 and apparently formed when highway construction cut off a meander bend in EFPC. The date this may have occurred is unknown. A culvert was placed at the western end of the wetland presumably in an attempt to drain it, but the culvert appears to be plugged. Soil samples taken on either side of the wetland show 0 ppm mercury. It is not clear whether sediment samples were taken in this wetland. Triangulation data show this wetland is probably uncontaminated. This is the only wetland in the EFPC floodplain with an obligate hydrophytic plant as dominant (duckweed or *Lemna* spp.). The drainage channel from the wetland may have been sampled accidentally during Phase 1a sampling (Tributary TC). Wetland 12 is on DOE property.

Wetland 13 - 0.25 ha (0.61 acres). Wetland 13 is located about halfway between Transects 7 and 8, coordinates E 6200 m (20336 ft) and E 6300 m (20664 ft), on the north bank of EFPC (Fig. 6.21). Although no sampling was conducted in this wetland, triangulation data indicate the wetland is probably uncontaminated. The wetland probably formed as a result of fluctuations in water level in EFPC due to the backwater effect from the Melton Hill and Watts Bar reservoirs. The wetland is roughly elliptic in shape with distinct narrow concentric bands of different vegetation types grading into a nonwetland as distance increases away from the site. In the center where ponding is most frequent there is virtually no vegetation. Next is a band of sedges, rushes, and other plants; a zone of shrubs; and a zone of bottomland hardwood trees. A small

distinct channel leading to EFPC from the wetland indicates that an attempt may have been made to drain this area in the past. A small spring also feeds into this wetland. Water levels may rise or fall rapidly depending on operations at the dams on Melton Hill and Watts Bar. Wetland 13 is on DOE property.

Wetland 14 - 0.0.35 ha (0.86 acres). Wetland 14 is located on the north bank of EFPC near Transect 7, coordinate E 6200 m (20336 ft) (Fig. 6.21). Transect 7 cuts across the western end of the wetland near the point where it meets EFPC. Sampling was conducted at two points in the wetland on either side of a small creek draining through the wetland. Mercury levels were 81 ppm on the south side of this channel and 0 on the north side. Despite the one measurement on mercury, triangulation data show the wetland is probably uncontaminated. This wetland is very similar to Wetland 13 in terms of origin, vegetation, and hydrology. Wetland 14 is located on the north bank about 170 m (560 ft) downstream from Wetland 13. There was evidence that beavers have been using this site. Wetland 14 is located on DOE property.

Wetland 15 - 0.03 ha (0.08 acres). Wetland 15 is located about halfway between Transects 6 and 7, coordinates E 6100 m (20008 ft) and E 6200 m (20336 ft) (Fig 6.21). Although no sampling was conducted in this wetland, triangulation data indicate the wetland is probably uncontaminated. This wetland is similar to Wetlands 13 and 14 in terms of origin and vegetation. Hydrology appears to be controlled by the backwater effect from the reservoirs; no creeks or springs flow into the wetland. The wetland is located on the south bank directly across EFPC from Wetland 14 on the south side of the creek. Wetland 15 is on DOE property.

Wetland 16 - 0.16 ha (0.39 acres). Wetland 16 is located on DOE property on the south side of EFPC along Transect 5, coordinate E 6000 m (19680 ft) (Fig. 6.21). The transect bisects the wetland. Only one sample was taken in this wetland, possibly because most of it was underwater at the time sampling was done. A sample was taken in the wetland at Station 08 that had a mercury value of 0 ppm. Triangulation data indicate the wetland is probably uncontaminated. This wetland is similar to Wetlands 13, 14, and 15 in terms of origin and vegetation; hydrology is controlled by the backwater effect from the reservoirs; no creeks or springs flow into the wetland. Wetland 16 is about 150 m (490 ft) downstream from Wetland 15.

Wetland 17 - 0.32 ha (0.80 acres). Wetland 17 is located on DOE property along Transect 4, coordinate E 5900 m (19352 ft), on the north bank of EFPC (Fig. 6.21). The transect bisects the wetland. Only one sample was taken in this wetland, possibly because most of it was underwater. One sample was taken at Station 06 that had a mercury value of 0 ppm. Triangulation data indicate this wetland is probably uncontaminated. This wetland is similar to

Wetlands 13, 14, 15, and 16 in terms of origin and vegetation. Hydrology is controlled by the backwater effect from the reservoirs; no creeks or springs flow into the wetland. It differs from the other wetlands only in the large size of the mudflat in this wetland in relation to the area occupied by other zones of vegetation. Wetland 17 is immediately across EFPC from Wetland 16.

Sixteen wetlands covering a total of 4.79 ha (11.82 acres) are located in the EFPC floodplain, representing three habitat types and varying degrees of disturbance by humans in addition to contamination from Y-12 and other sources. Because of their position in the landscape many of these wetlands have been exposed to mercury contamination. Triangulation data indicate that as many as 2.6 ha (6.43 acres) of wetlands have been contaminated with mercury and possibly other COCs. Many terrestrial and aquatic animals have been observed using these areas during different seasons of the year. The assessment of the possible long-term effects of contamination on the functions and values of wetlands and other important ecological resources and habitats continues.

Color-coded habitat maps. These color plates (Figs. 6.21 through 6.37) show EFPC Sections 1-17 and the color-coded distributions of terrestrial and aquatic habitats in each successive reach of EFPC from the confluence with Poplar Creek to the Y-12 Plant boundary. They also show the locations of ecological sampling sites 1 through 6. The list on the following page presents the figure number and title for each plate; the plates themselves are identified by section number.

6.2.1.5 Estimated future exposures

In the absence of remedial activities in the EFPC floodplain and additional controls on sources of contaminants released into the water column from the Y-12 Plant, the nature, extent, and distribution of contaminants in the EFPC ecosystem are not expected to change dramatically. Future exposures and risks are also handled in Section 6.4.2. Changes in the ecosystem as a result of remediation in the EFPC floodplain will be analyzed in detail in the FS/EIS.

6.2.2 Description of Exposure Routes

6.2.2.1 Exposure models

Contaminant exposure routes for the two major contaminant sources—the Y-12 Plant including sediment exposure and EFPC floodplain soils including soil ingestion—are summarized in Figs. 6.38 and 6.39. As described in Sects. 6.2.1.1 and 6.2.1.3, exposure of aquatic biota

Color Plates (Figs. 6.21 - 6.37)

Fig. 6.21. Section 1—Distribution of terrestrial and aquatic habitats in EFPC from confluence with Poplar Creek to Y-12 Plant boundary.

Fig. 6.22. Section 2—Distribution of terrestrial and aquatic habitats in EFPC from confluence with Poplar Creek to Y-12 Plant boundary.

Fig. 6.23. Section 3—Distribution of terrestrial and aquatic habitats in EFPC from confluence with Poplar Creek to Y-12 Plant boundary.

Fig. 6.24. Section 4—Distribution of terrestrial and aquatic habitats in EFPC from confluence with Poplar Creek to Y-12 Plant boundary.

Fig. 6.25. Section 5—Distribution of terrestrial and aquatic habitats in EFPC from confluence with Poplar Creek to Y-12 Plant boundary.

Fig. 6.26. Section 6—Distribution of terrestrial and aquatic habitats in EFPC from confluence with Poplar Creek to Y-12 Plant boundary.

Fig. 6.27. Section 7—Distribution of terrestrial and aquatic habitats in EFPC from confluence with Poplar Creek to Y-12 Plant boundary.

Fig. 6.28. Section 8—Distribution of terrestrial and aquatic habitats in EFPC from confluence with Poplar Creek to Y-12 Plant boundary.

Fig. 6.29. Section 9—Distribution of terrestrial and aquatic habitats in EFPC from confluence with Poplar Creek to Y-12 Plant boundary.

Fig. 6.30. Section 10—Distribution of terrestrial and aquatic habitats in EFPC from confluence with Poplar Creek to Y-12 Plant boundary.

Fig. 6.31. Section 11 —Distribution of terrestrial and aquatic habitats in EFPC from confluence with Poplar Creek to Y-12 boundary.

Fig. 6.32. Section 12—Distribution of terrestrial and aquatic habitats in EFPC from confluence with Poplar Creek to Y-12 Plant boundary.

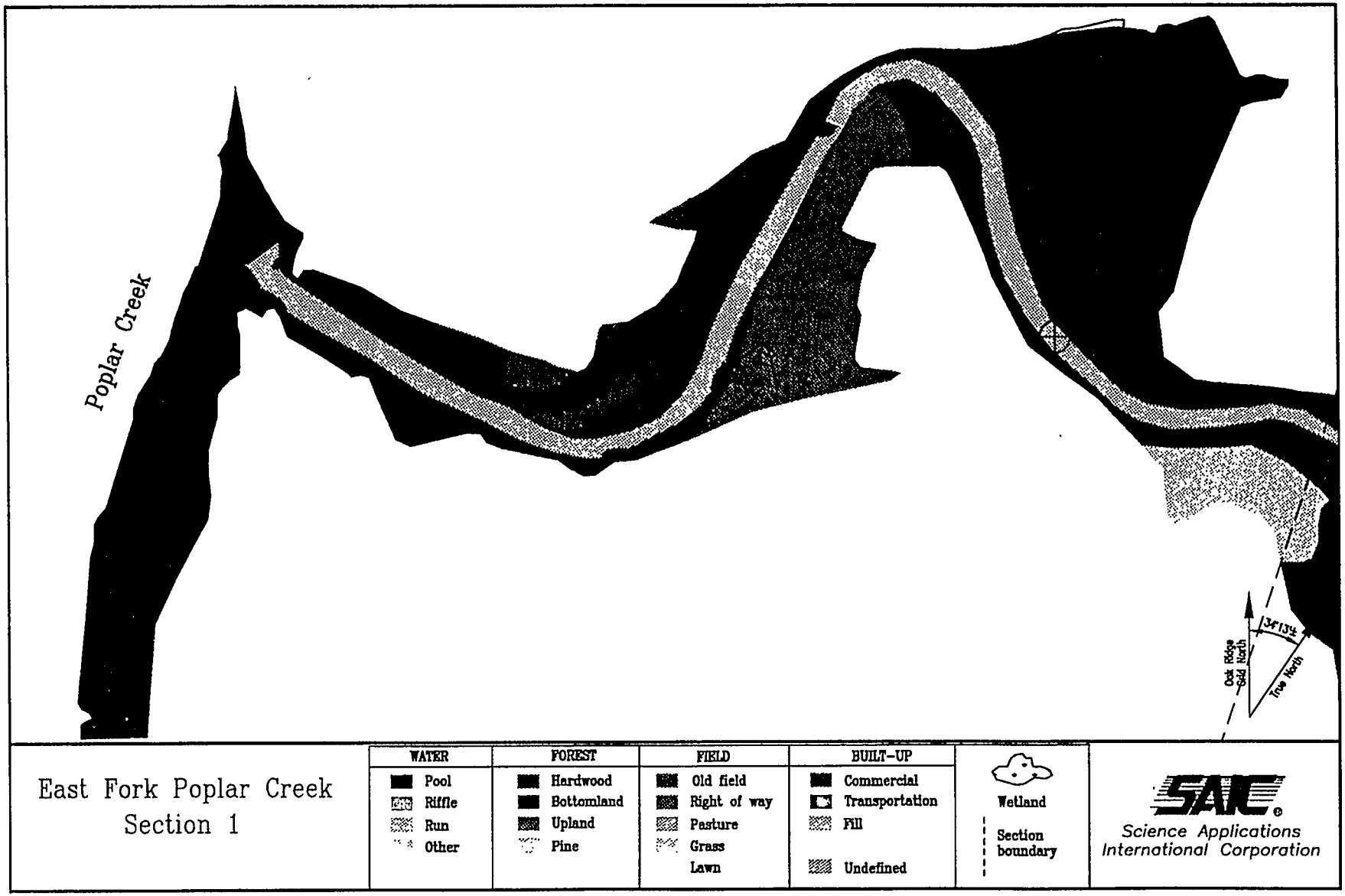
Fig. 6.33. Section 13—Distribution of terrestrial and aquatic habitats in EFPC from confluence with Poplar Creek to Y-12 Plant boundary.

Fig. 6.34. Section 14—Distribution of terrestrial and aquatic habitats in EFPC from confluence with Poplar Creek to Y-12 Plant boundary.

Fig. 6.35. Section 15—Distribution of terrestrial and aquatic habitats in EFPC from confluence with Poplar Creek to Y-12 Plant boundary.

Fig. 6.36. Section 16—Distribution of terrestrial and aquatic habitats in EFPC from confluence with Poplar Creek to Y-12 Plant boundary.

Fig. 6.37. Section 17—Distribution of terrestrial and aquatic habitats in EFPC from confluence with Poplar Creek to Y-12 Plant boundary.



East Fork Poplar Creek
Section 1

WATER	FOREST	FIELD	BUILT-UP
Pool	Hardwood	Old field	Commercial
Riffle	Bottomland	Right of way	Transportation
Run	Upland	Pasture	Fill
Other	Pine	Grass	
		Lawn	Undefined

Wetland

Section boundary

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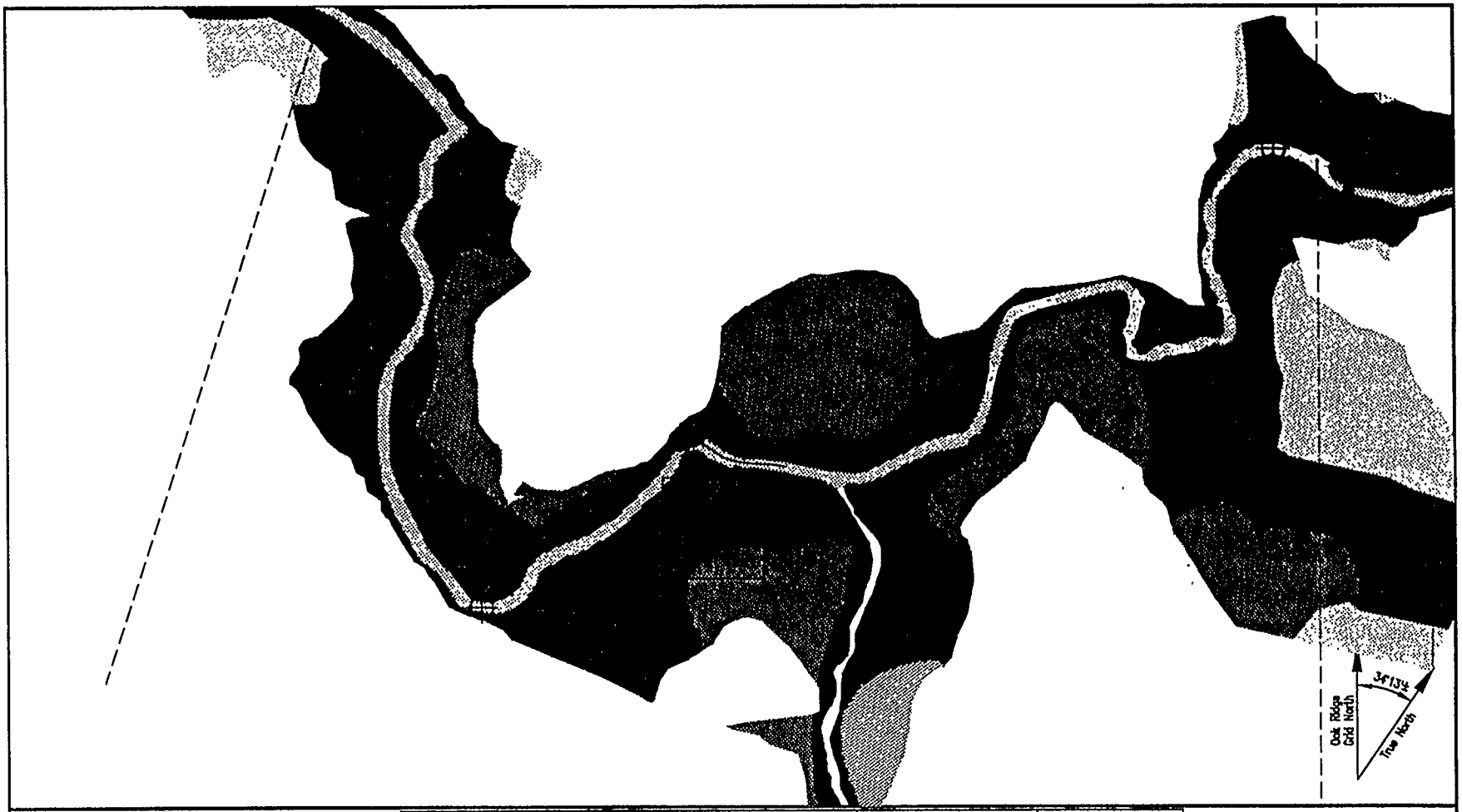

East Fork Poplar Creek
Section 2

WATER	FOREST	FIELD	BUILT-UP
Pool	Hardwood	Old field	Commercial
Riffle	Bottomland	Right of way	Transportation
Run	Upland	Pasture	Fill
Other	Pine	Grass	
		Lawn	Undefined

Wetland

Section boundary

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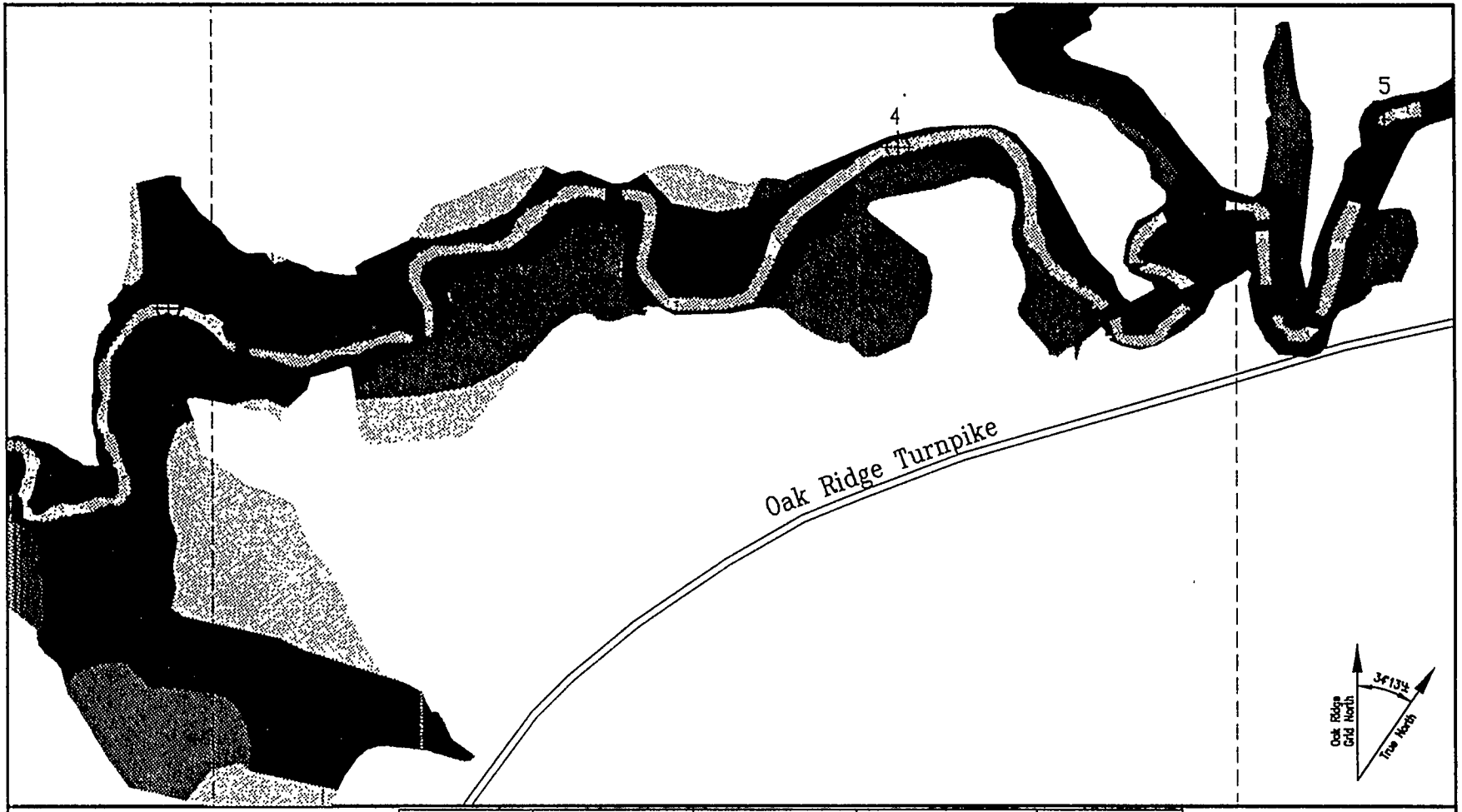

East Fork Poplar Creek
Section 3

WATER	FOREST	FIELD	BUILT-UP
Pool	Hardwood	Old field	Commercial
Riffle	Bottomland	Right of way	Transportation
Run	Upland	Pasture	Fill
Other	Pine	Grass	
		Lawn	Undefined

Wetland

Section boundary

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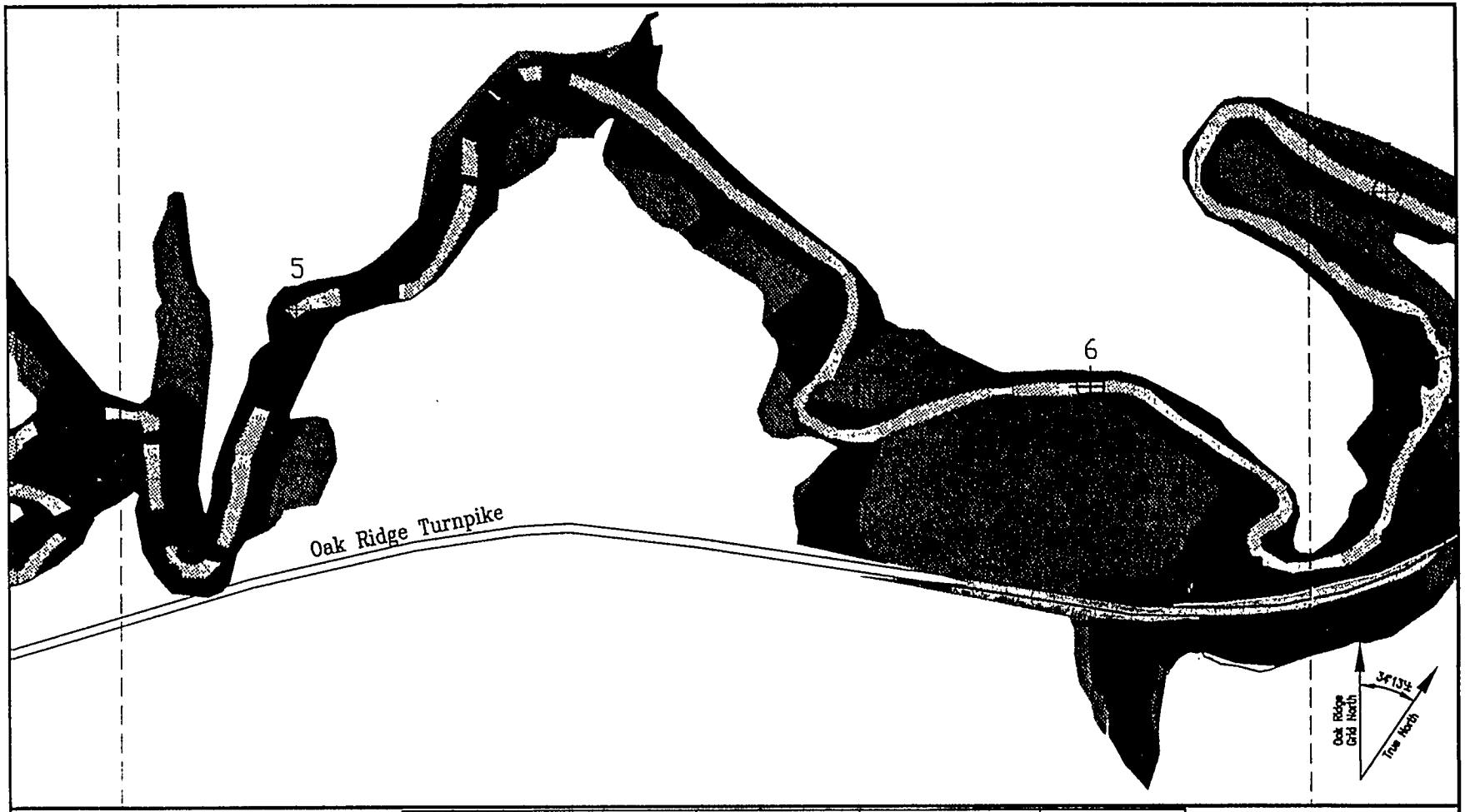

East Fork Poplar Creek
Section 4

WATER	FOREST	FIELD	BUILT-UP
Pool	Hardwood	Old field	Commercial
Riffle	Bottomland	Right of way	Transportation
Run	Upland	Pasture	Fill
Other	Pine	Grass	
		Lawn	Undefined

Wetland

Section boundary

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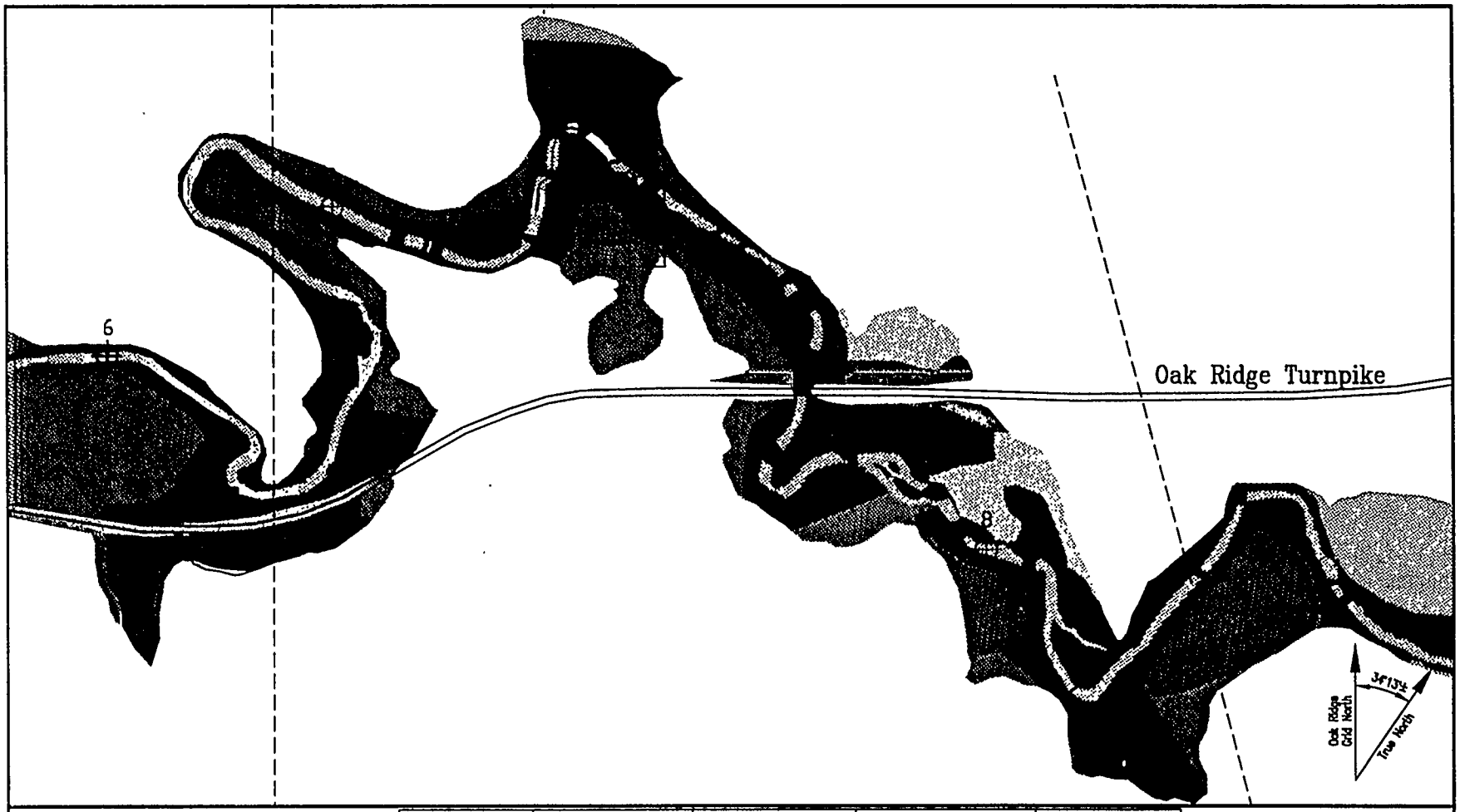

East Fork Poplar Creek
Section 5

WATER	FOREST	FIELD	BUILT-UP
Pool	Hardwood	Old field	Commercial
Riffle	Bottomland	Right of way	Transportation
Run	Upland	Pasture	Fill
Other	Pine	Grass	
		Lawn	Undefined

Wetland

Section boundary

SAIC®
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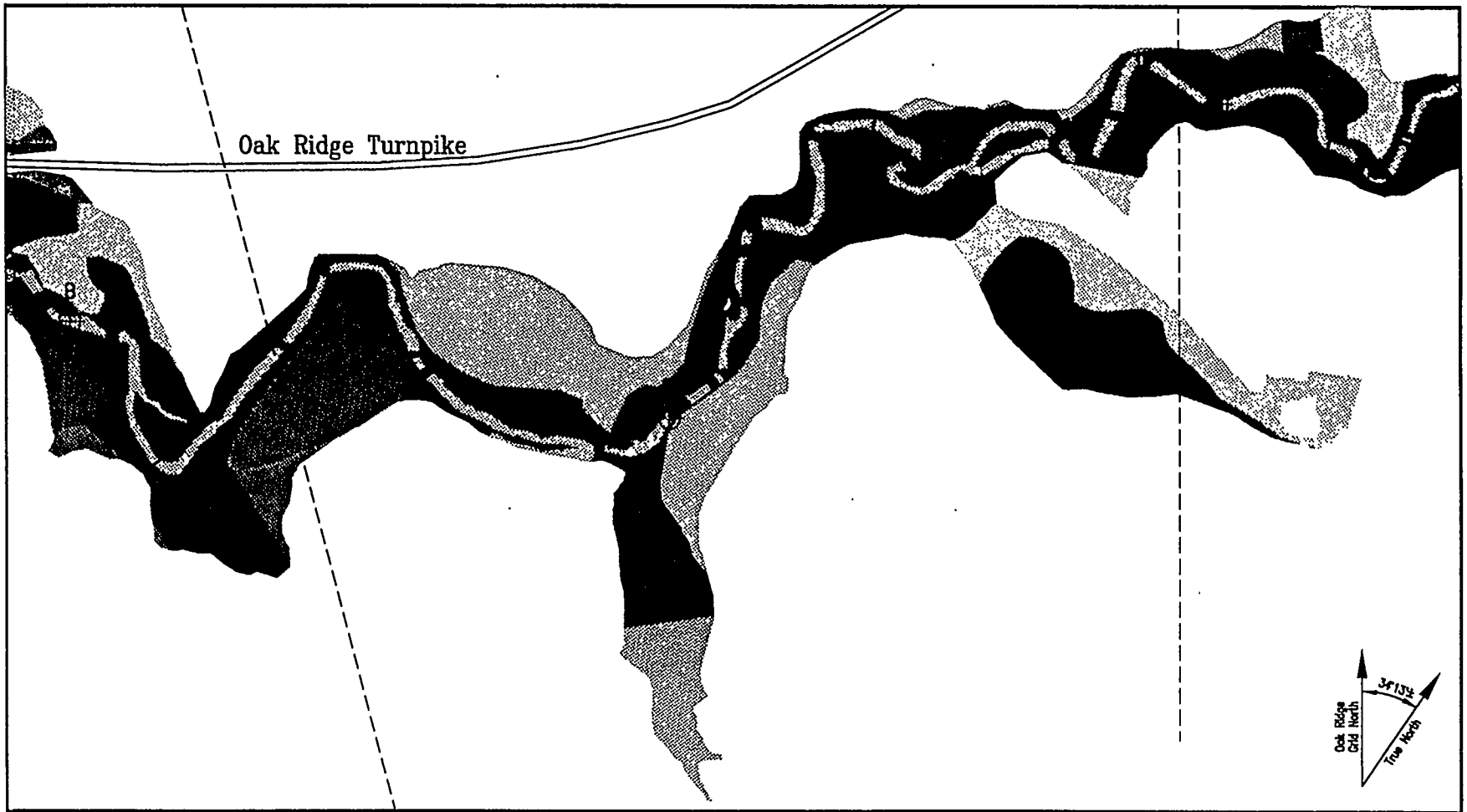

East Fork Poplar Creek
Section 6

WATER	FOREST	FIELD	BUILT-UP
Pool	Hardwood	Old field	Commercial
Riffle	Bottomland	Right of way	Transportation
Run	Upland	Pasture	Fill
Other	Pine	Grass	
		Lawn	Undefined

Wetland

Section boundary

SAIC®
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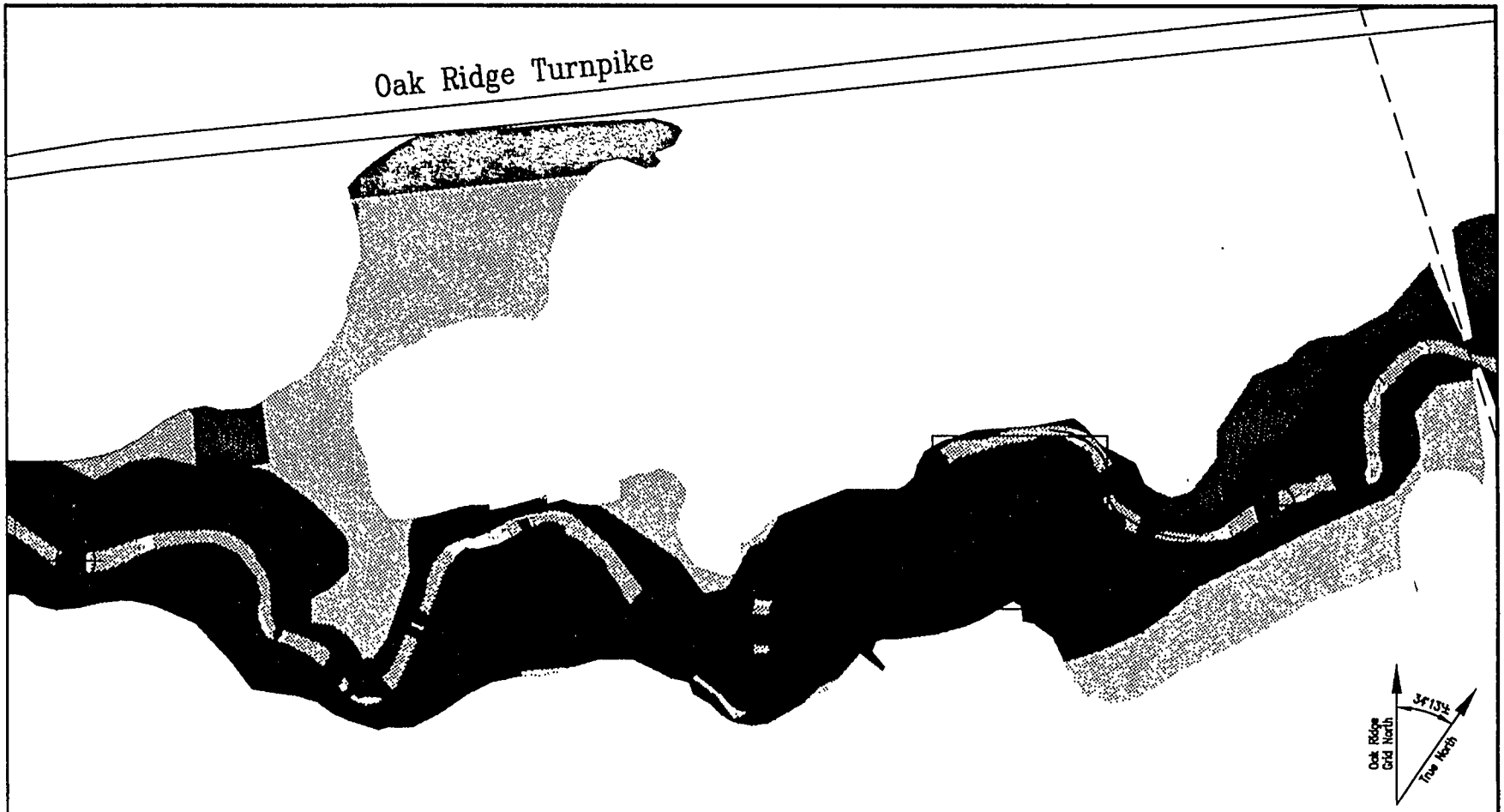

East Fork Poplar Creek
Section 7

WATER	FOREST	FIELD	BUILT-UP
Pool	Hardwood	Old field	Commercial
Riffle	Bottomland	Right of way	Transportation
Run	Upland	Pasture	Fill
Other	Pine	Grass	
		Lawn	Undefined

Wetland

Section boundary

SAIC®
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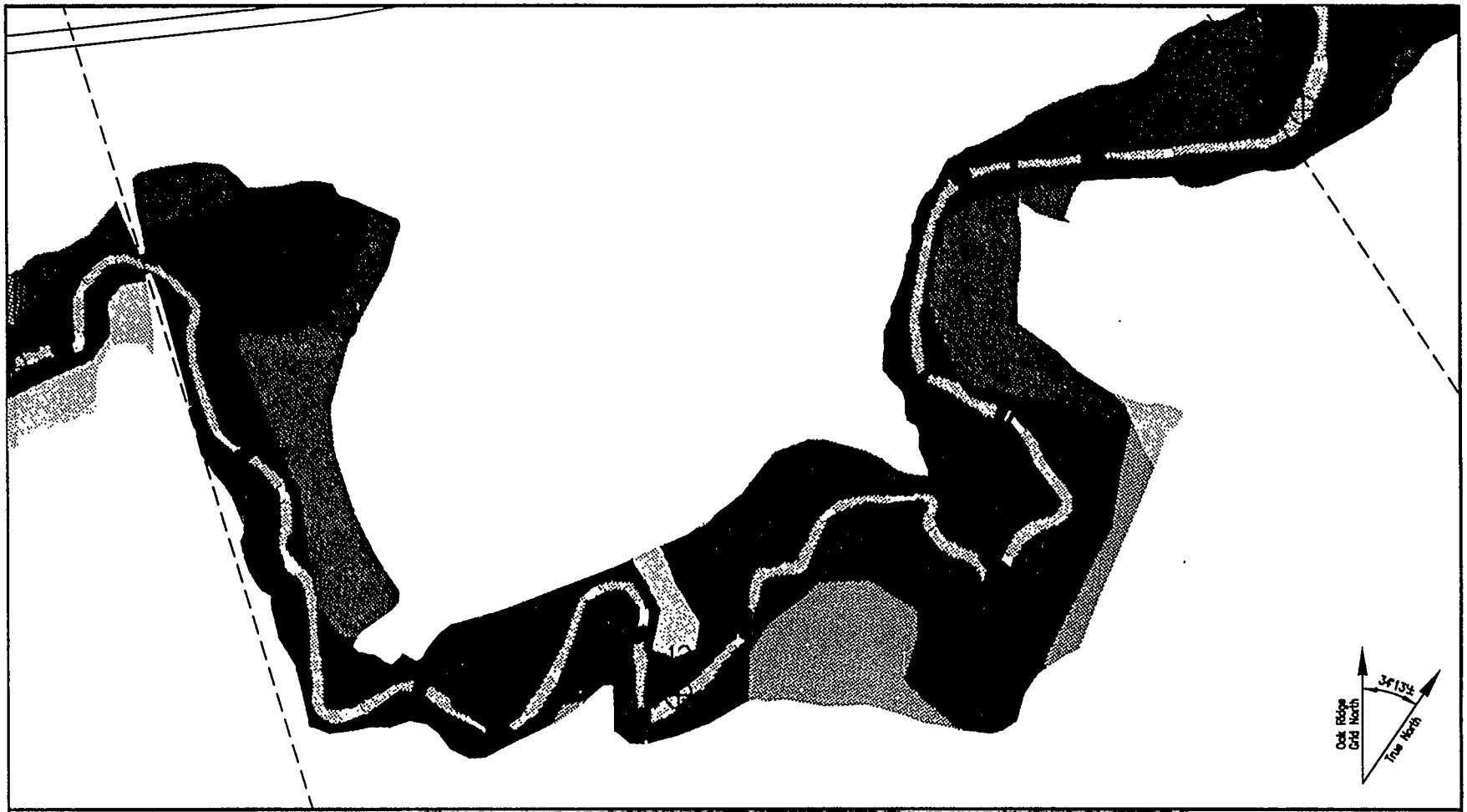

East Fork Poplar Creek
Section 8

WATER	FOREST	FIELD	BUILT-UP
Pool	Hardwood	Old field	Commercial
Riffle	Bottomland	Right of way	Transportation
Run	Upland	Pasture	Fill
Other	Pine	Grass	
		Lawn	Undefined

Wetland

Section boundary

SAIC®
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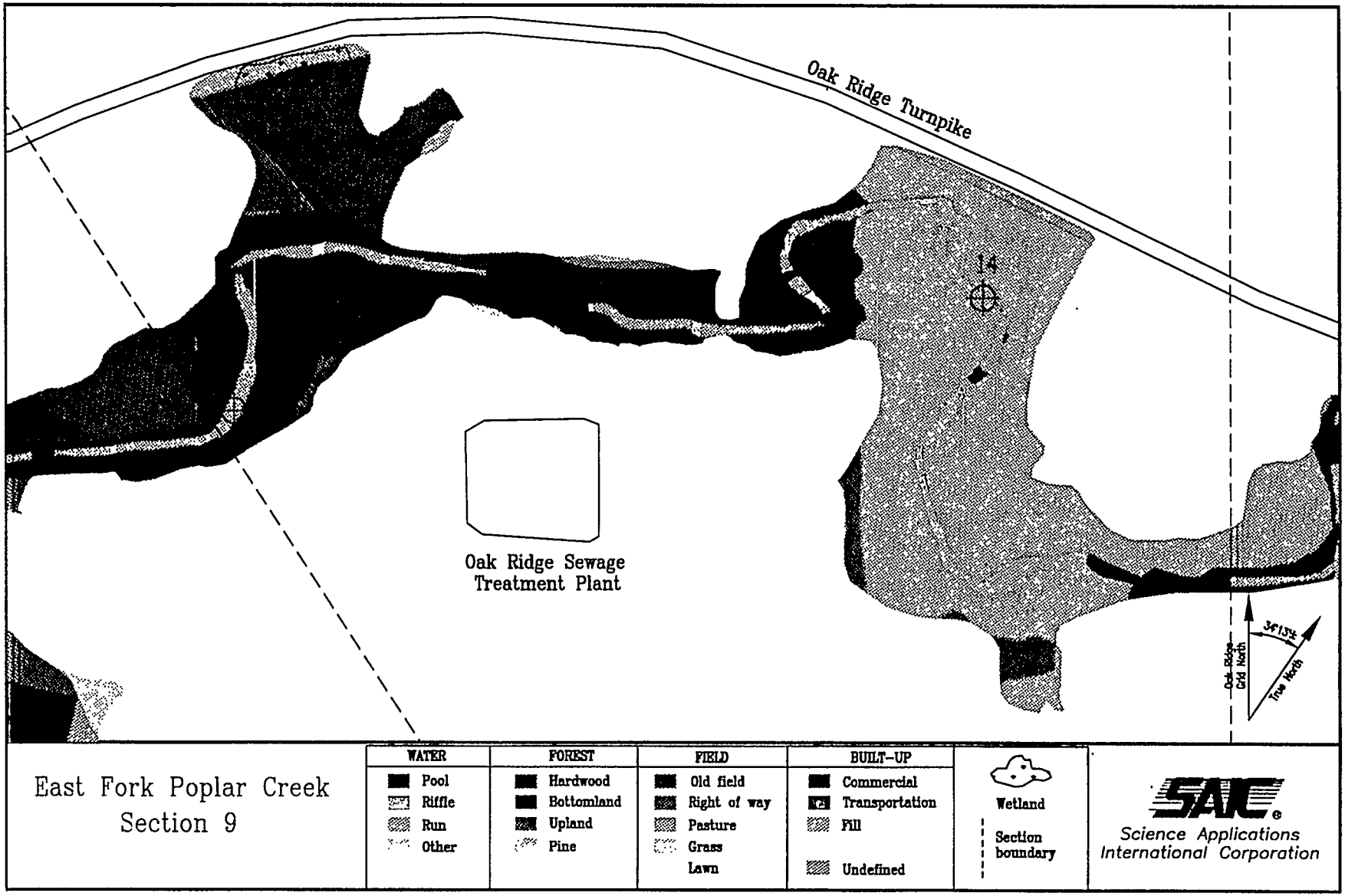
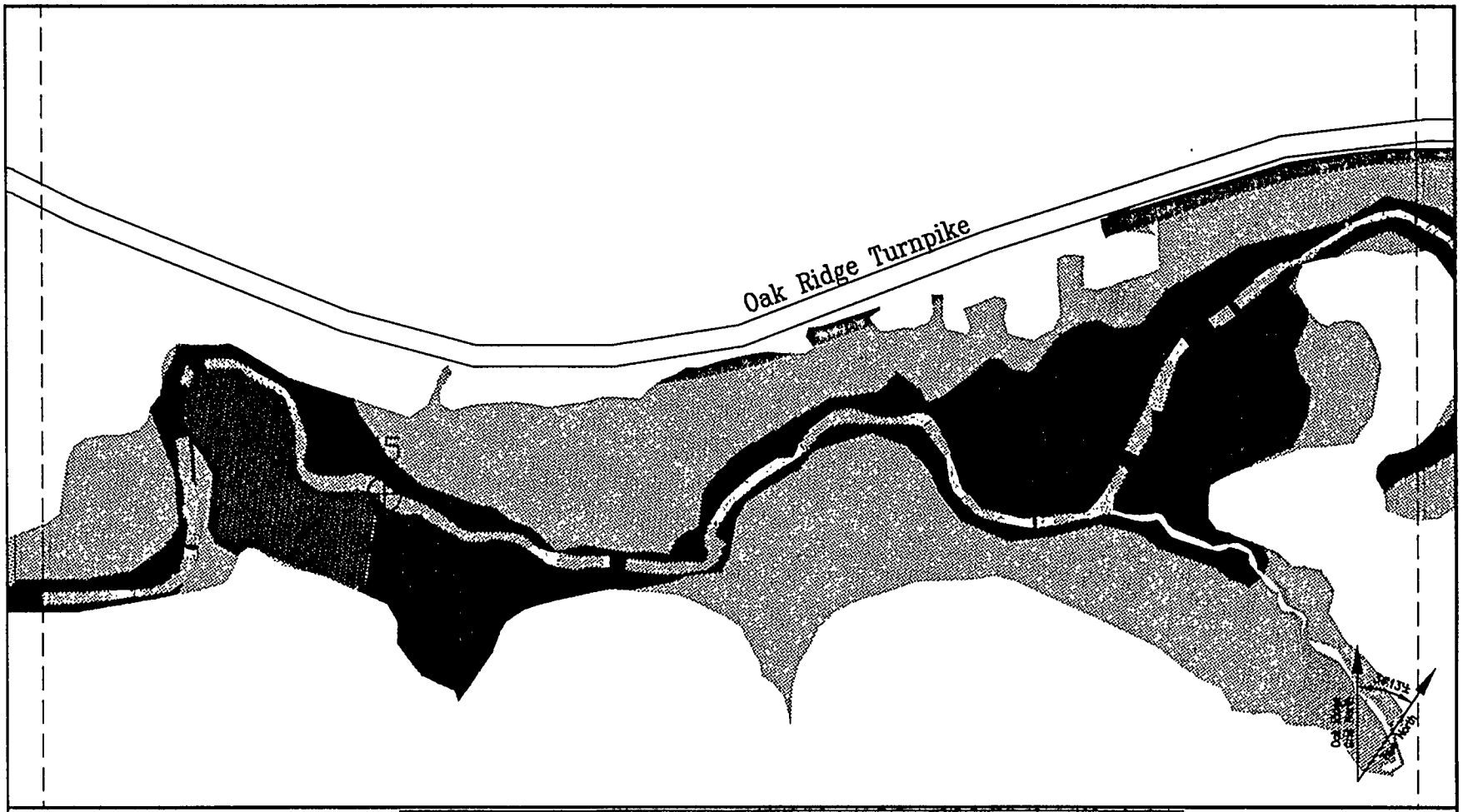

Oak Ridge Turnpike
14
Oak Ridge Sewage
Treatment Plant
34°13½
Oak Ridge
Grid North
True North
East Fork Poplar Creek
Section 9
WATER
Pool
Riffle
Run
Other
FOREST
Hardwood
Bottomland
Upland
Pine
FIELD
Old field
Right of way
Pasture
Grass
Lawn
BUILT-UP
Commercial
Transportation
Fill
Undefined
Wetland
Section
boundary
SAIC
Science Applications
International Corporation



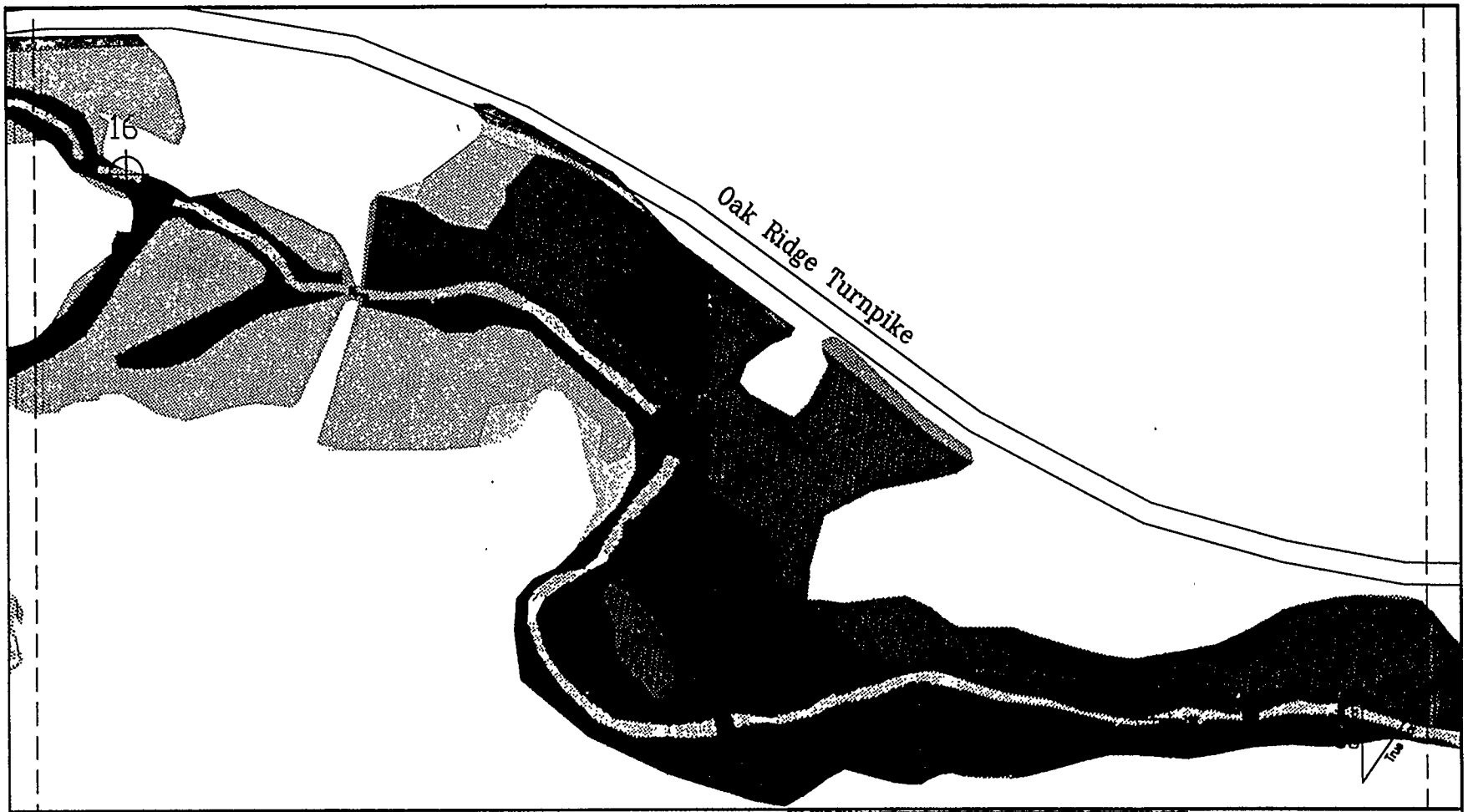
East Fork Poplar Creek
Section 10

WATER	FOREST	FIELD	BUILT-UP
Pool	Hardwood	Old field	Commercial
Riffle	Bottomland	Right of way	Transportation
Run	Upland	Pasture	Fill
Other	Pine	Grass	
		Lawn	Undefined

Wetland

Section boundary

Science Applications International Corporation



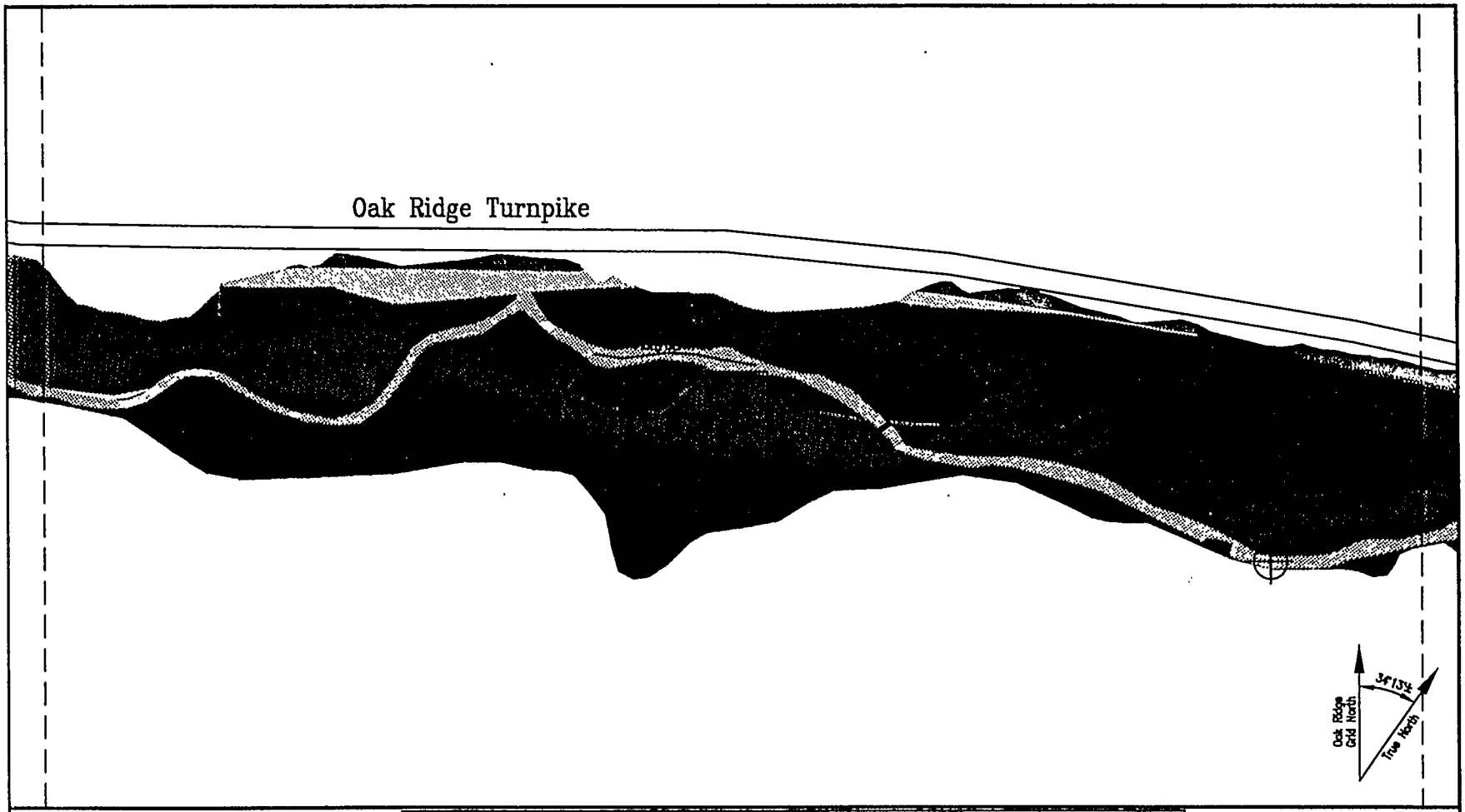
East Fork Poplar Creek
Section 11

WATER	FOREST	FIELD	BUILT-UP
Pool	Hardwood	Old field	Commercial
Riffle	Bottomland	Right of way	Transportation
Run	Upland	Pasture	Fill
Other	Pine	Grass	
		Lawn	Undefined

Wetland

Section boundary

Science Applications International Corporation



East Fork Poplar Creek
Section 12

WATER	FOREST	FIELD	BUILT-UP
Pool	Hardwood	Old field	Commercial
Riffle	Bottomland	Right of way	Transportation
Run	Upland	Pasture	Fill
Other	Pine	Grass	
		Lawn	Undefined

Wetland

Section boundary

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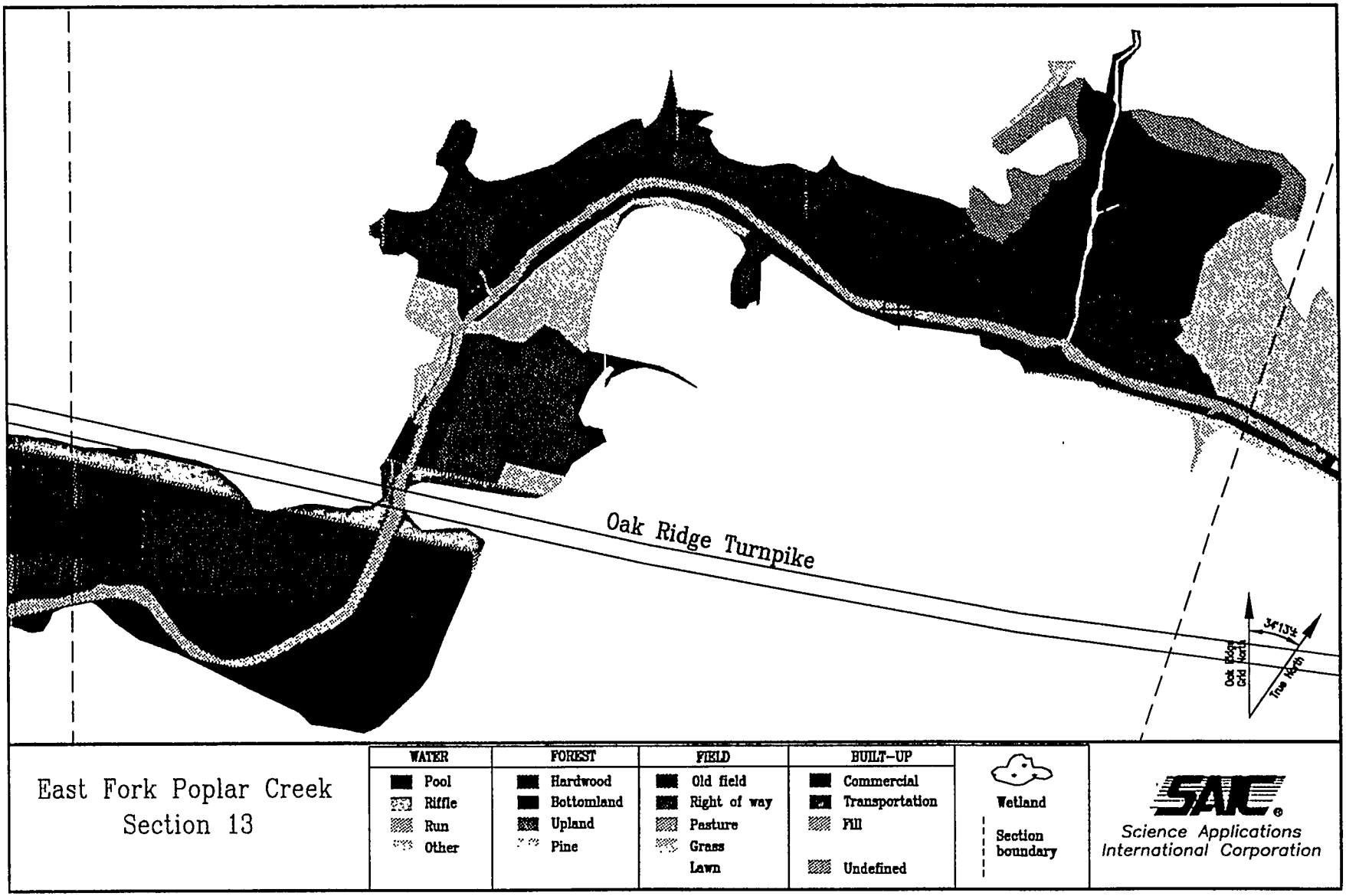


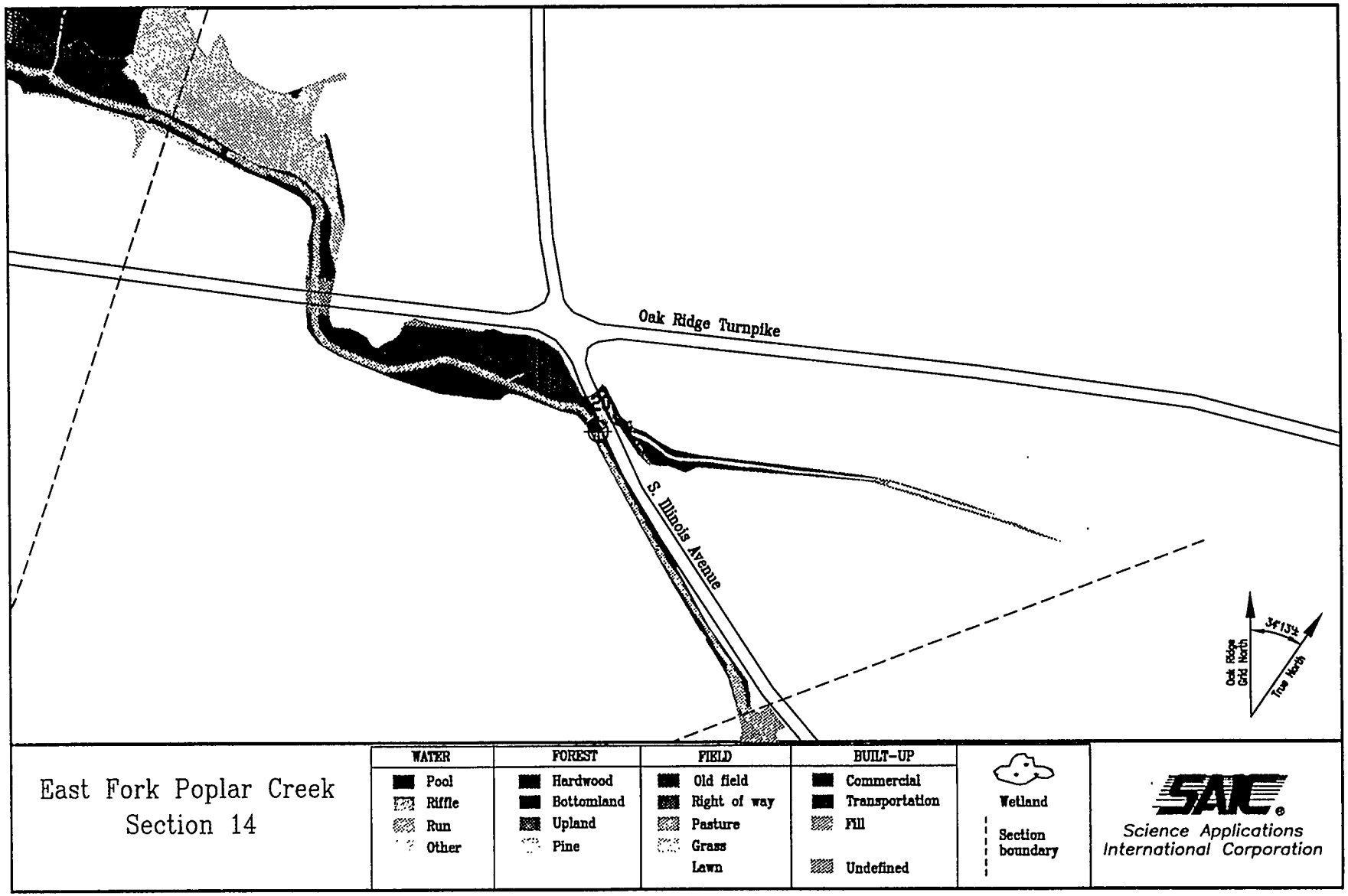
East Fork Poplar Creek
Section 13

WATER	FOREST	FIELD	BUILT–UP
Pool	Hardwood	Old field	Commercial
Riffle	Bottomland	Right of way	Transportation
Run	Upland	Pasture	Fill
Other	Pine	Grass	
		Lawn	Undefined

Wetland

Section boundary

SAIC®
Science Applications International Corporation



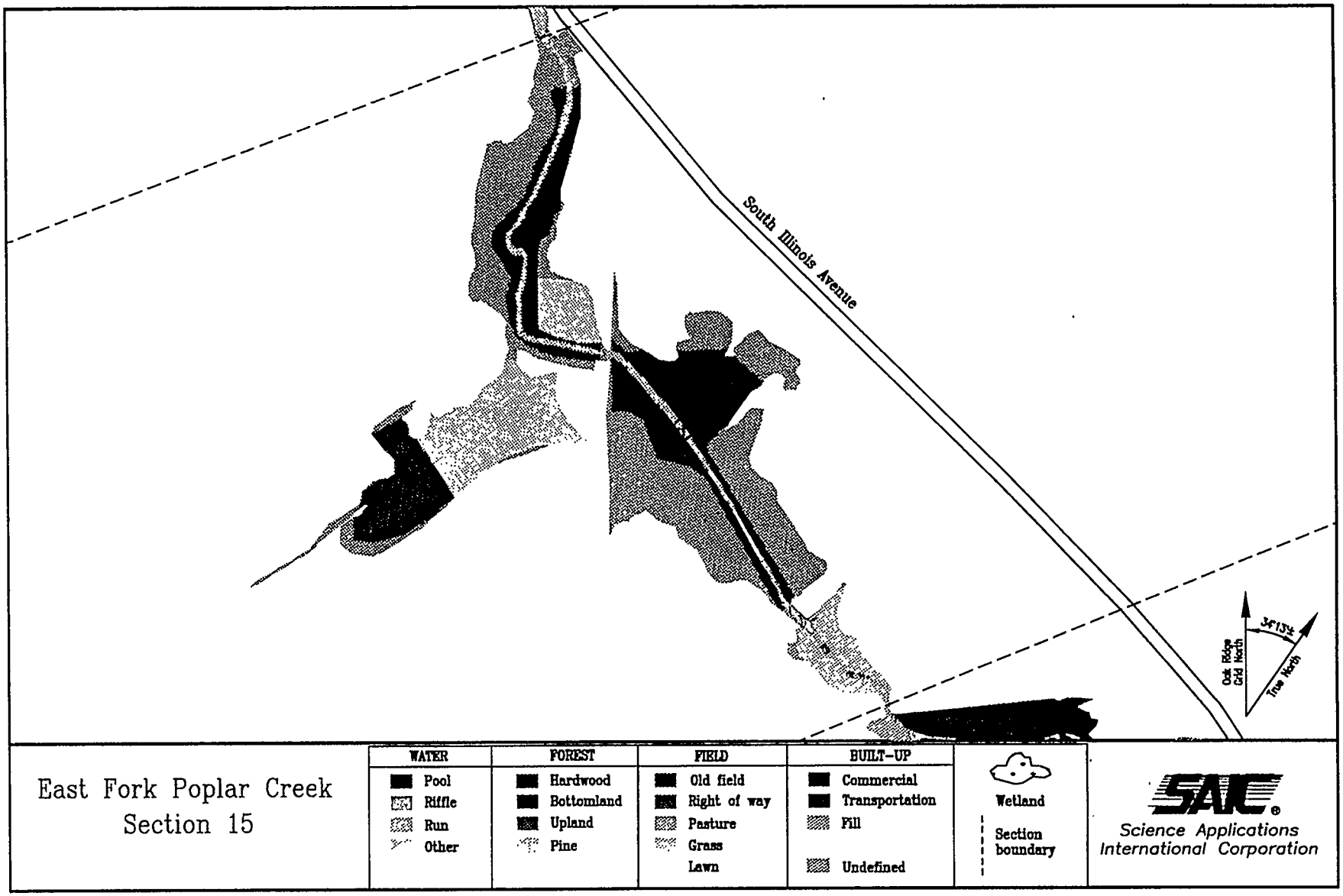
East Fork Poplar Creek
Section 14

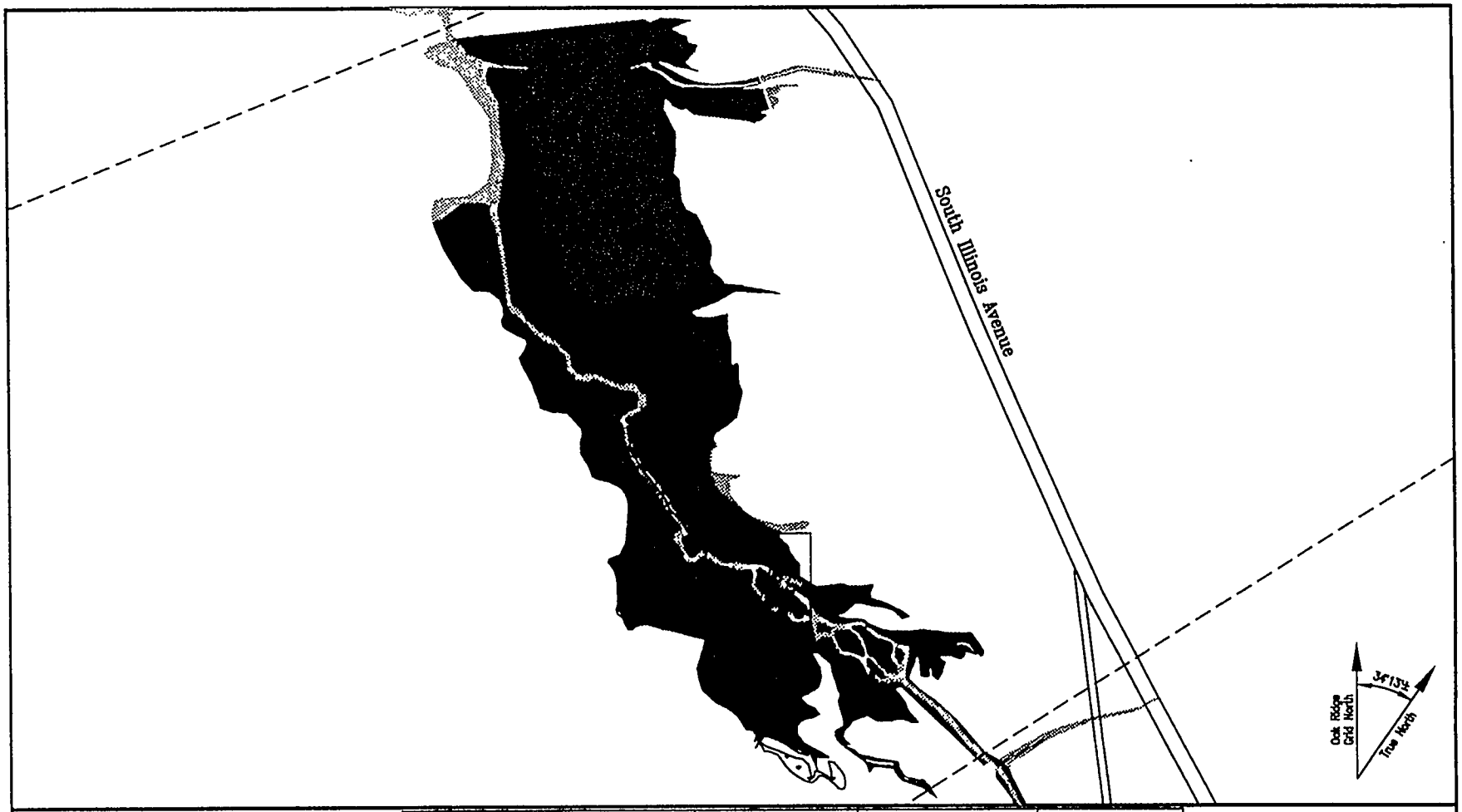
WATER	FOREST	FIELD	BUILT-UP
Pool	Hardwood	Old field	Commercial
Riffle	Bottomland	Right of way	Transportation
Run	Upland	Pasture	Fill
Other	Pine	Grass	
		Lawn	Undefined

Wetland

Section boundary

Science Applications International Corporation


South Illinois Avenue
3°13'E
Oak Ridge Grid North
True North
East Fork Poplar Creek
Section 15
WATER
Pool
Riffle
Run
Other
FOREST
Hardwood
Bottomland
Upland
Pine
FIELD
Old field
Right of way
Pasture
Grass
Lawn
BUILT-UP
Commercial
Transportation
Fill
Undefined
Wetland
Section boundary
SAIC
Science Applications International Corporation



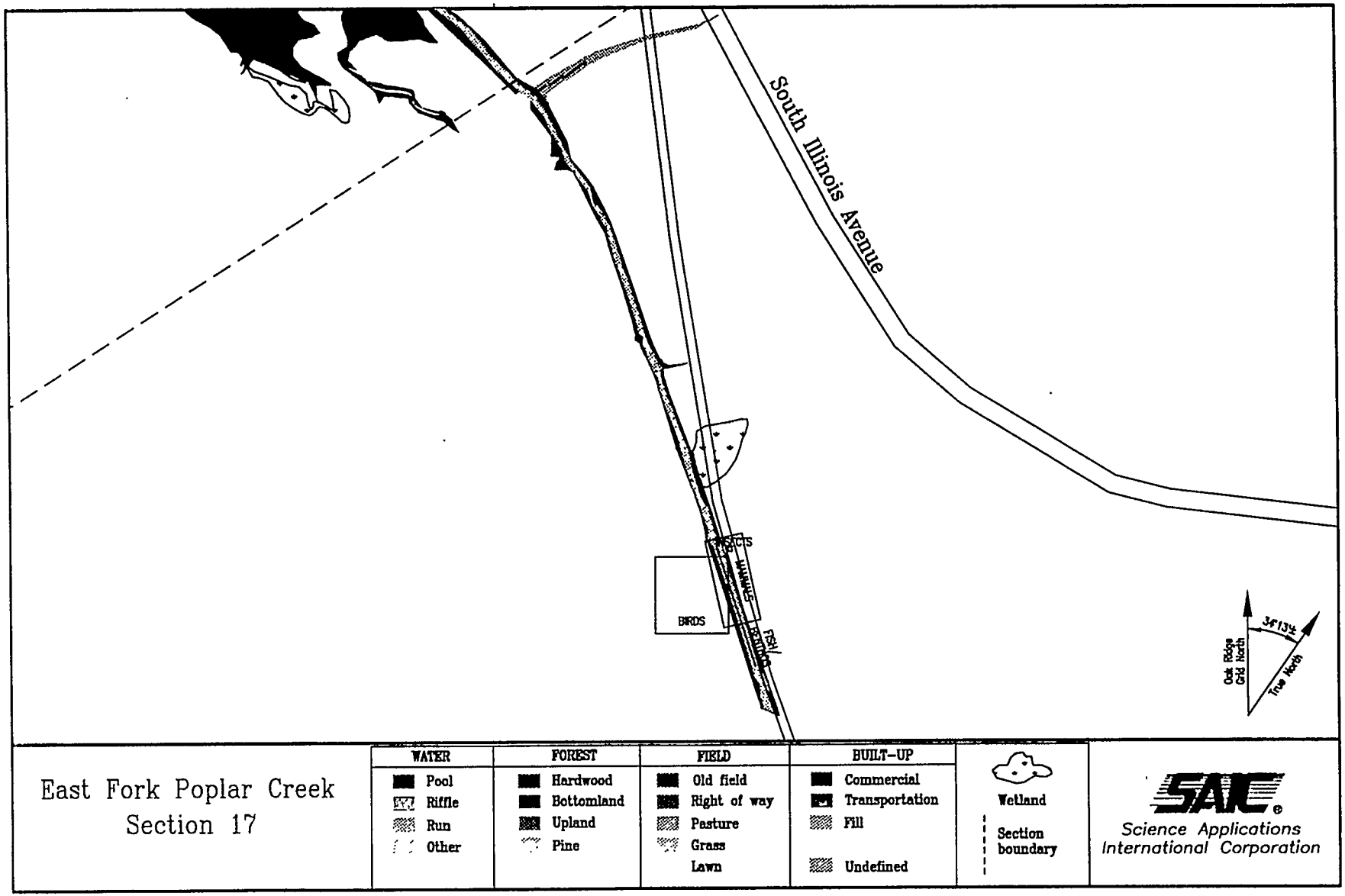
East Fork Poplar Creek
Section 16

WATER	FOREST	FIELD	BUILT-UP
Pool	Hardwood	Old field	Commercial
Riffle	Bottomland	Right of way	Transportation
Run	Upland	Pasture	Fill
Other	Pine	Grass	
		Lawn	Undefined

Wetland

Section boundary

Science Applications International Corporation



East Fork Poplar Creek
Section 17

WATER	FOREST	FIELD	BUILT-UP
Pool	Hardwood	Old field	Commercial
Riffle	Bottomland	Right of way	Transportation
Run	Upland	Pasture	Fill
Other	Pine	Grass	
		Lawn	Undefined

Wetland

Section boundary

Science Applications International Corporation

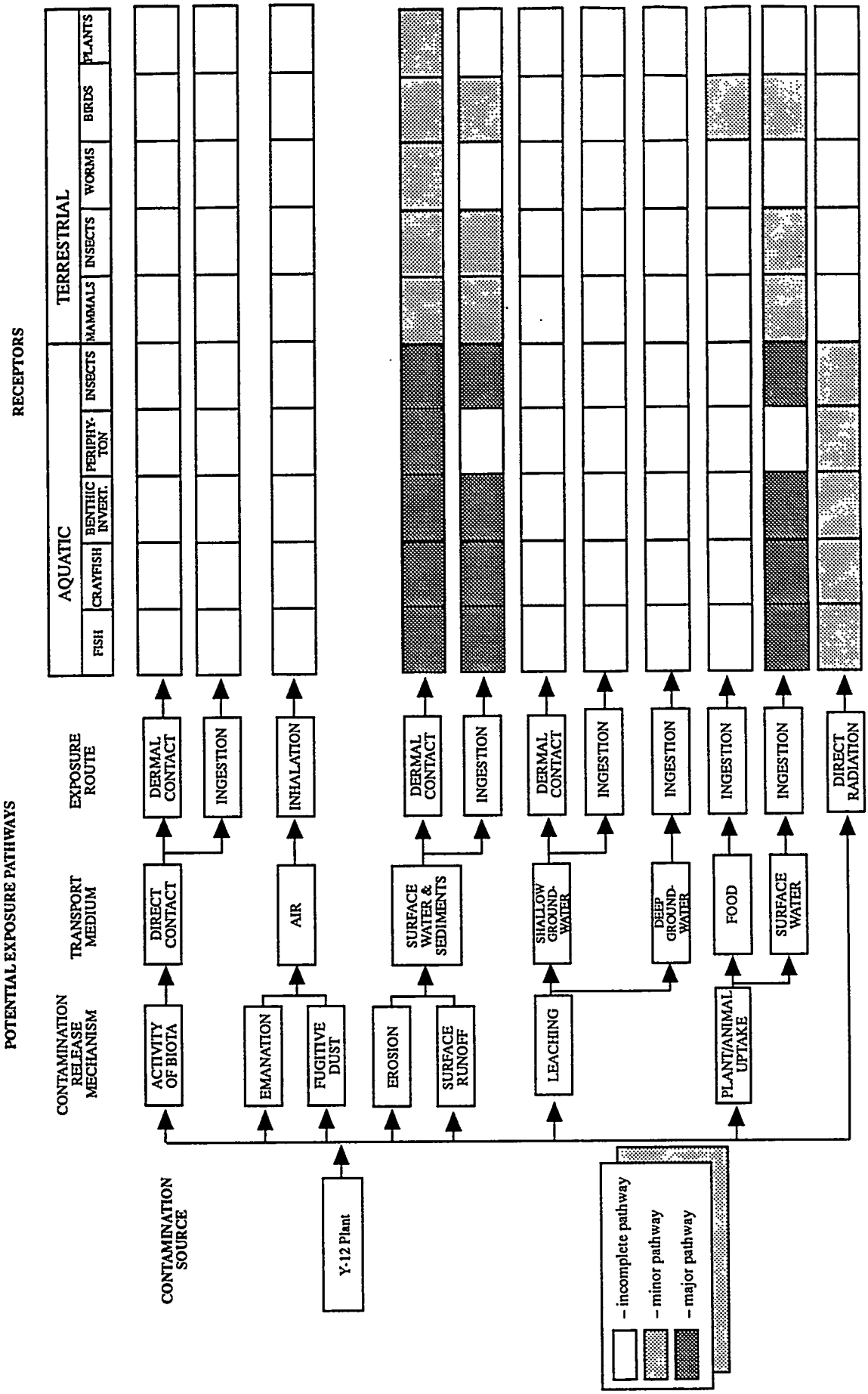


Figure 6.38. Conceptual site model of exposure pathways for Y-12 Plant contaminants.

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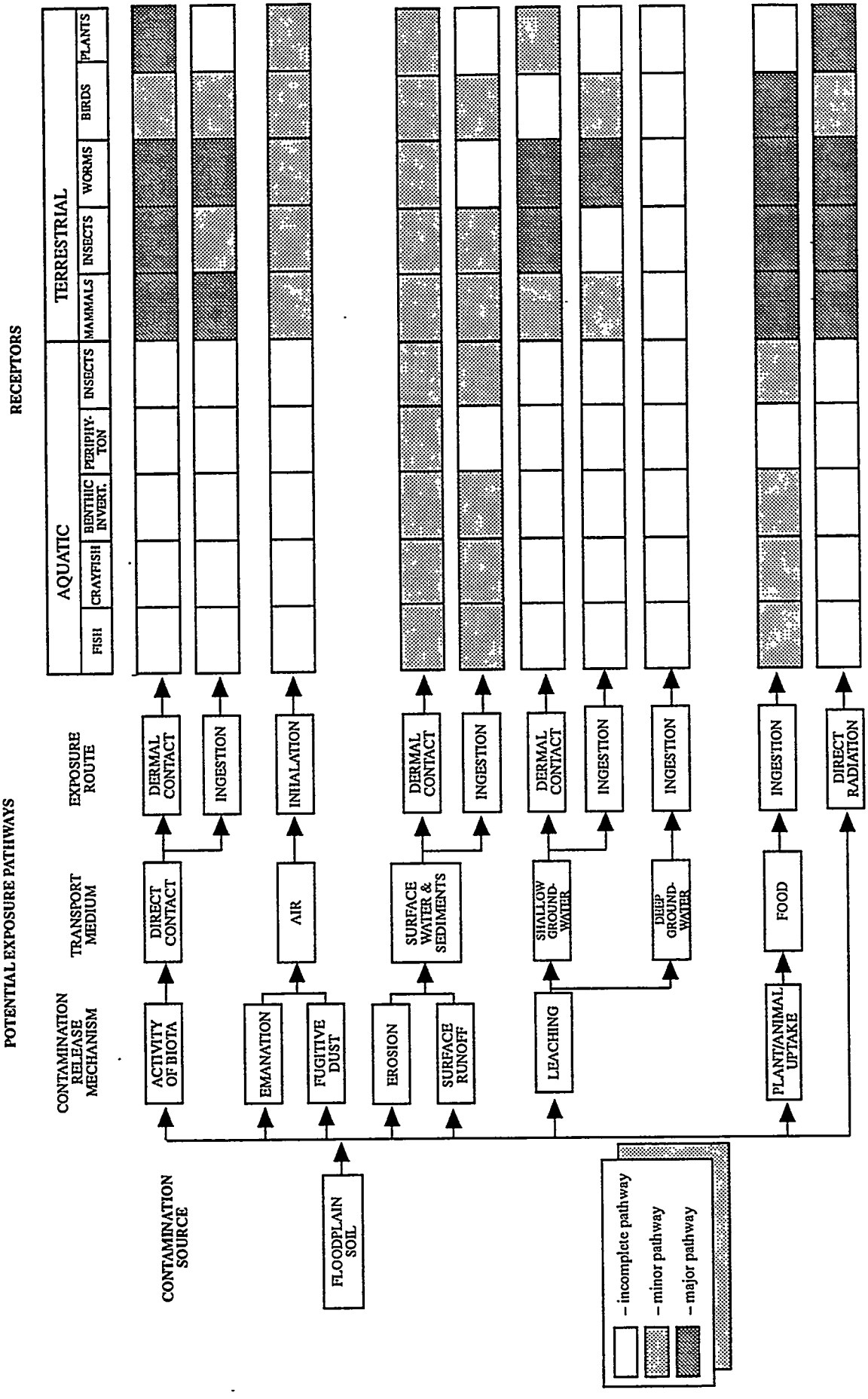

Figure 6.39. Conceptual site model of exposure pathways for EFPC floodplain soil contaminants.

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to contaminants from the Y-12 Plant occurs largely by way of surface water. A simplified food web showing the potential movement of contaminants from surface water through the biotic receptors used in this study is shown in Fig. 6.40. Exposure to contaminants in soils is very limited for aquatic receptors. The potential flow of contaminants from floodplain soils to terrestrial biotic receptors in this study is diagrammed in Fig. 6.41.

Y-12 Plant. Exposure pathways for contaminants originating in the Y-12 Plant, whether direct or via the food web, depend primarily on surface water exiting Lake Reality. Although birds may move into and out of the Y-12 Plant site, biota of the other species sampled for body burden analysis appear not to migrate in significant numbers from the Y-12 Plant into the downstream reaches of EFPC or its floodplain. Therefore, neither direct body contact with nor ingestion of contaminants within the Y-12 Plant appear to be significant exposure routes for EFPC biota. Similarly, exposure to airborne contaminants is not expected to be significant, and deep groundwater is not accessible to biota. Shallow groundwater in the Y-12 Plant can contribute to exposures in the EFPC floodplain only by its discharge to surface water.

Aquatic biota are exposed to contaminants almost continuously by direct contact with surface water, and most are exposed by ingestion. Crayfish and benthic macro-invertebrates may be exposed to instream sediments by direct contact to various degrees. Benthic macroinvertebrates and periphyton sampled in this study were taken from riffle areas, in which sediment deposition is not extensive. Therefore, direct exposure to sediments should have been limited for these indicators. Crayfish were collected from the entire 100-m (330-ft) sampling reach, which included areas having more extensive sediment deposition. Bottom-feeding biota, such as stonerollers and benthic macroinvertebrates, are likely to ingest sediments as they feed. Suspended sediments may also be ingested to a minor degree along with ingested water.

Terrestrial mammals that forage in or near water, such as raccoons, are exposed both topically and by ingestion to contaminants in surface water and sediments. The small mammal indicators used in this study, as well as earthworms and flying insects of terrestrial origin, should not have been exposed to surface water and sediments to a significant degree. Piscivorous birds may be exposed to both surface water and sediment to degrees depending on their feeding habits. For example, wading birds would be more exposed to sediments by both direct contact and ingestion than would kingfishers. Ducks are exposed to surface water by direct contact and ingestion, and may also ingest significant amounts of sediment as they feed. In contrast, songbirds are less likely to be exposed to contaminants in surface water and sediments. Terrestrial plants in low-lying areas near EFPC may occasionally be exposed to surface water during floods. These flood events are infrequent, but because the transport of sediment-bound

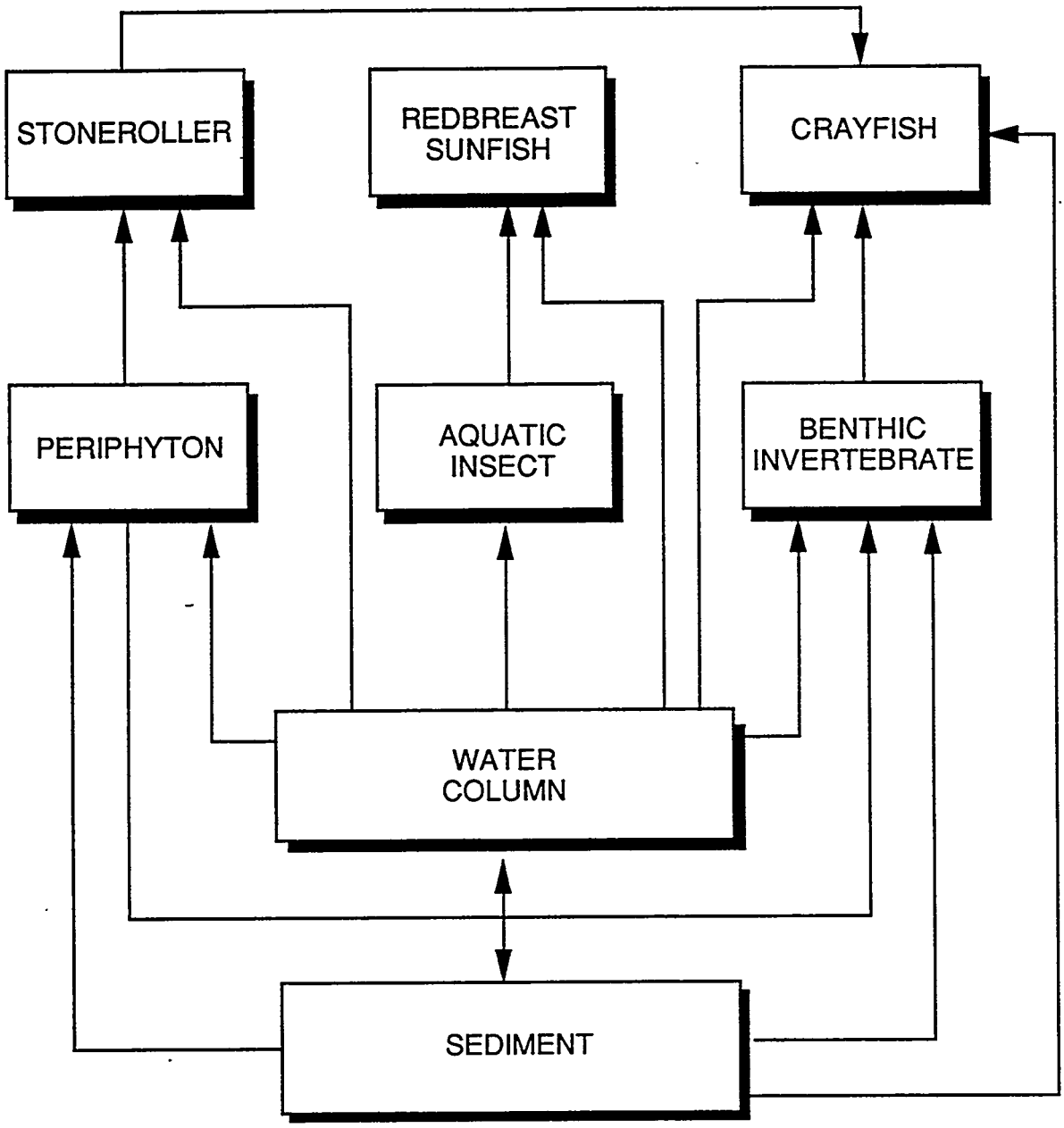

Fig. 6.40. Food web relationships of aquatic biota sampled for EFPC ecological risk assessment.

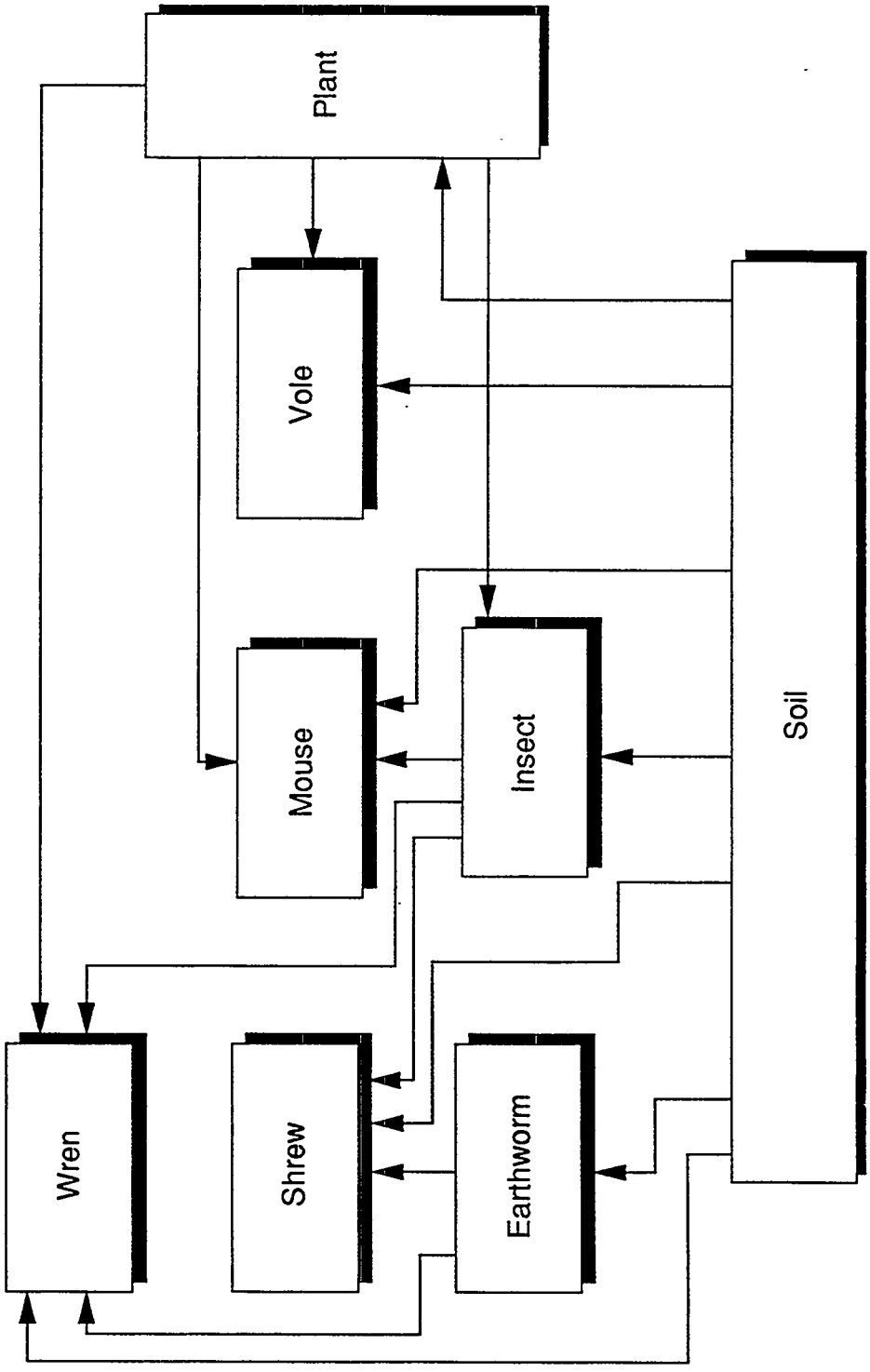

Fig. 6.41. Contaminant movement from floodplan soils to receptors.

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contaminants is highest during floods, exposure to contaminants may be higher during flood conditions.

The most significant pathway for exposure to contaminants from the Y-12 Plant source is the food web. Birds in the EFPC floodplain may feed within the Y-12 Plant boundaries, and thereby be exposed to contaminants in their food. Of greater consequence, mercury appears to enter the food web on the Y-12 Plant site, exiting Lake Reality in surface water as methylmercury incorporated in plankton and other small biota (Appendix Q). Methylmercury passes through the food web to all the aquatic indicator organisms. Piscivorous terrestrial mammals and birds are exposed by ingestion of contaminated fish, crayfish, and other invertebrates such as molluscs. These pathways may result in significant impacts to terrestrial populations as contaminants are bioaccumulated through the food web.

Sediment exposures via pore water. Instream sediments may be an important source of ecological exposure to contaminants arising from the Y-12 Plant (Fig. 6.38) or from the EFPC floodplain (Fig. 6.39). Benthic macroinvertebrates live within the sediment layer and are exposed by dermal absorption and ingestion, and crayfish and stonerollers may be exposed by incidental ingestion of sediment during feeding. All of these taxa showed elevated body burdens of contaminants such as mercury and PCBs (Sect. 6.2.3.2).

A number of methods have been proposed for the assessment of sediment toxicity (Adams et al. 1992). The equilibrium partitioning (EP) method is a predictive method that allows risk projections based on concentrations of contaminants in instream sediment. Contaminant exposure in sediment typically occurs by leaching of contaminants into pore water. Contaminant concentrations in pore water and in the particulate or solid phase are assumed to be at chemical equilibrium. Therefore, the concentration of contaminant in one phase can be predicted if the concentration in the other phase and the equilibrium coefficient are known. The advantage of this method is that partition coefficients can be predicted from octanol-water partition coefficients (K_{ow}) for many organic compounds, so site-specific data are not required. Application of the EP method to biotic exposures in EFPC sediments is discussed in the following subsection.

For inorganic contaminants, site-specific partition coefficients (K_D) must be determined. Values of K_D for the binding of mercury to EFPC sediments were calculated from results of the mesocosm study reported in Appendix Q. Equilibrium concentrations of mercury in pore water are given by the equation:

$$C_{water} = C_{sediment} \times 1000 / K_D$$

where

C_{water} = Concentration in water (μg/L)
$C_{sediment}$ = Concentration in sediment (mg/kg)
K_D = Partition coefficient (L/kg)
1000 = Conversion factor, μg to mg.

The mean K_D value for total mercury from the mesocosm study was 8.85×10^5 L/kg, and the mean and maximum observed sediment mercury concentrations were 14.9 mg/kg and 95.6 mg/kg, respectively (Table 3.18). The corresponding calculated equilibrium pore water mercury concentrations are 0.017 μg/L and 0.11 μg/L.

Nonpolar organic compounds bind to the organic carbon portion of soils and sediments, with an avidity (described by the constant K_{oc}) that is related to the total organic carbon content (TOC) of the particulates. Thus, a site-specific K_D is not necessary to calculate equilibrium partitioning for organic compounds when TOC is specified. The relationship used is the identity:

$$K_D = K_{oc} \times f_{oc}$$

where f_{oc} is the fraction of the soil represented by TOC of the particulates. For nonpolar organic contaminants, $C_{sediment}$ is given in μg/kg, and

$$C_{water} = C_{sediment} / (K_{oc} \times f_{oc})$$

For PCBs, we use the approximation

$$K_{oc} = 7.8 \times 10^5 \text{ L/kg (Lyman et al. 1990)}$$

Values for TOC in EFPC sediments are not available, but soil TOC in the EFPC floodplain (a potential source of instream sediments) ranged from 1% to 11%. It is standard practice in exposure assessment to calculate a most likely exposure (MLE) from the mean exposure value and a reasonable maximum exposure (RME) using the 95% upper confidence limit of mean of the exposure distribution. However, sediment pore water exposure is inversely related to sediment TOC, which means that lower TOCs result in higher concentrations in pore water. Therefore, for the calculation of sediment exposure, the 95% lower confidence limit of the mean

TOC will be used. The mean TOC for upper horizon soils was 3.33%, with a 95% lower confidence limit of 2.15%. The maximum observed PCB concentration in a sediment composite was 420 μg/kg (Table 3.18). Using this value and the soil TOC range in the equilibrium partitioning formula,

$$C_{water} = C_{sediment} / (K_{oc} \times f_{oc})$$
$$= 420\ \mu g/kg / (7.8 \times 10^5\ L/kg \times [0.0215 \text{ to } 0.0333])$$
$$= 0.016 \text{ to } 0.025\ \mu g/L.$$

Sediments used in the mesocosm study (Appendix Q) had a range of 6.1 to 7.5% carbon, which computes to a sediment pore water concentration range of 0.007 μg/L to 0.009 μg/L. For PCBs, ambient water quality criterion (AWQC) = 0.001 μg/L. The calculated values exceed this number by quotients of 7 to more than 25 for the maximum sediment PCB concentration observed. Therefore, sediments containing more than about 50 μg PCB/kg (depending on organic carbon content) may pose a risk to sediment biota.

EFPC floodplain soils. Soil is not expected to provide exposure to contaminants by either direct contact or air transport to any aquatic biota. Similarly, aquatic biota have no access to deep groundwater. Shallow groundwater emerging from springs and seeps contributes to surface water, but it is not considered to be a medium of exposure for aquatic biota. Erosion and surface runoff transport contaminants from floodplain soils into EFPC, allowing topical exposure of all aquatic biota and exposure by ingestion for all the aquatic indicators except periphyton. Exposure via erosion and surface runoff is transient, occurring predominantly during periods of heavy rainfall. Uptake of contaminants from EFPC floodplain soils into aquatic plants, mammals, and invertebrates is a minor source of exposure. Fish, crayfish, and, to some extent, benthic macroinvertebrates may feed on plant detritus or insects of terrestrial origin that are incidentally deposited in the creek. Although larger animals that are predominantly aquatic may consume small animals and birds that could be exposed to terrestrial contaminants, these animals were not chosen as indicators in this study, and risk analysis about them would, therefore, be limited to indirect measures (e.g., food).

Terrestrial mammals are exposed to contaminants in soil both topically and by ingestion. Feeding habits and other aspects of lifestyle determine the significance of these exposures. For example, shrews prey on earthworms, which may have exterior contamination from adhering soils and contain ingested soil. Therefore, shrews ingest much more soil than voles, whose diet is predominantly vegetation. Some insects of terrestrial origin spend their entire life on or in the

soil. Flying insects used as indicators in this study include beetles whose immature forms are burrowing grubs, as well as wasps and moths whose immature forms may have little contact with soil. Therefore, the extent of exposure for insect indicator groups depends on the composition of each sample. Earthworms are highly exposed to contaminants in soil by both dermal contact and ingestion. They are a likely source of contaminants for shrews, some birds, and other small animals. Birds are exposed to various degrees of soil contaminants. They may take dust baths in EFPC floodplain soil or ingest soil inadvertently as they feed or intentionally for grit. The degree of exposure depends on how extensively their home range overlaps the floodplain and whether they are transient or permanent residents. Plants are highly exposed to contaminants in EFPC soils by root uptake and by surface deposition of airborne dust or sediment during flooding.

Potential exposure by ingestion of soil can be estimated by using standard exposure assessment methodology (EPA 1989c) and site-specific data. Feeding studies have shown that absorption of mercury and other metals by laboratory mice from contaminated soil taken from the EFPC floodplain caused no discernible toxicological effects during a 20-month exposure (Revis et al. 1989b). In this study, the fraction of soil in the diet was 0.05; higher exposures under natural conditions are very unlikely. Therefore, it is concluded that incidental ingestion of soil does not present a risk to terrestrial biota in the EFPC floodplain.

The absorption of organic contaminants from soil may be higher and is assessed in the following subsection. An estimate of contaminant intake by soil ingestion can be made by methods analogous to those found in Sect. 5 (Human Health Assessment). The soil exposure quotient is calculated according to the formula:

$$CI = (C_{soil} \times 10^{-6}\ kg/mg \times SI \times FI \times ABS)/(BW \times 10^{-3}\ kg/g)$$

where

- CI = Contaminant intake (mg/kg/d)
- C_{soil} = Contaminant concentration in soil (mg contaminant/kg soil)
- SI = Soil intake (100 mg soil/d)
- FI = Fraction of soil from contaminated area (1.0)
- ABS = Fraction of ingested dose absorbed (1.0)
- BW = Body weight (25 g)

Therefore, $CI = 0.004 \times C_{soil}$

Justification and comments on variables:

SI: Incidental ingestion of soil by small mammals is likely to be variable, depending on feeding habits. Birds may ingest soil intentionally for grit to help grind their food. For this calculation, an arbitrary value of 100 mg/d (~0.4% of body weight) is used. This value is probably overly conservative for mice, but it may be an underestimate for shrews, which burrow and forage in fossorial zone.

FI: It is assumed here that the home range of the subject animal is restricted to the contaminated area of the EFPC floodplain (FI=1). That assumption is probably reasonable for mice and shrews.

ABS: The absorption factor may depend on the medium in which the contaminant is contained. Typically, the absorption factor of organic chemicals is assumed to be 1, whereas it is usually lower for inorganic chemicals. If the medium used to determine the comparison dose was the same as the exposure medium, no correction for absorption should be made.

BW: Body weight of mice is typically ~25-40 g. Variability in body weight is likely to be compensated by a similar variability in soil ingestion.

Contaminated soil carried by wind may be inhaled by terrestrial birds and animals. Animals may inhale contaminants volatilized from the soil, and plants may take them up by transpiration or by direct absorption. This pathway appears to be minor, but chronic low-level exposure to airborne mercury and organics may occur.

Soil contaminants washed into EFPC by surface runoff or erosion may be dissolved or may be transported as suspended sediments. As described for contaminants from the Y-12 Plant, this route provides minor exposures from both dermal contact and ingestion for some mammals and birds and from direct contact and root uptake for plants, but probably insignificant or no exposures for earthworms and insects of terrestrial origin.

Leaching of soil contaminants into shallow groundwater is potentially a significant release mechanism leading to exposure of terrestrial biota. Shallow groundwater may be transported to EFPC, where it mixes with surface water. It may also emerge as seeps or springs, where terrestrial mammals, insects, and birds may be exposed by both dermal contact and ingestion. The significance of this pathway to small mammals depends on the source of water in their diet.

Deep groundwater is not accessible to terrestrial biota and does not constitute a complete exposure route.

Biota. A major source of exposure to EFPC floodplain soil contaminants for terrestrial biota is the food web. Shrews prey on earthworms, which are highly exposed to soil contaminants, and on insects and grubs, which are exposed by contact with soil and by ingestion of plant material, other invertebrates, and detritus. Mice and voles have a more mixed diet and, therefore, receive an intermediate exposure to soil contaminants.

Insects may feed on other insects, plants, or detritus, all of which may be contaminated by uptake from soil. The degree of exposure depends on the feeding habits of the insects. Similarly, earthworms ingest detritus that may be contaminated. Birds are exposed by ingestion of plant materials, insects, and earthworms. Most plants are not carnivorous and do not ingest contaminants; rather, plants may accumulate contaminants via root uptake.

6.2.3 Quantification of Exposure

Overall, both historical and current studies of bioaccumulation showed decreased body burdens of mercury, PCBs/pesticides, or both in stonerollers, sunfish, crayfish, earthworms, and certain terrestrial insects with increasing distance downstream from the Y-12 Plant, and higher body burdens than organisms from presumably uncontaminated reference sites. Current body burdens are generally lower than those from the late 1980s, possibly as a result of prior remediation activities. The notable exceptions to these overall patterns were the high present body burdens of mercury in redbreast sunfish from Site 3, 6.4 km downstream from Lake Reality, and the increasing body burden of PCBs in sunfish from Site 3 and sites downstream. Table 6.15 lists the common and scientific names of the fish species examined during these historical studies.

6.2.3.1 Historical internal and external bioaccumulation studies

1982 mercury concentration study - aquatic portion. A study was conducted in May 1982 to measure mercury concentrations in sediment and fish as well as mosses, liverwort, and pasture grasses (discussed separately in this section) at various sites in EFPC and Bear Creek (Van Winkle et al. 1984). Bluegill sunfish (*Lepomis macrochirus*) and a few specimens of largemouth bass and white bass (*Morone chrysops*) were collected by electrofishing at EFK 22.8, EFK 22.7, EFK 13.4, and EFK 2.1. Skinless fillets from the axial muscle were analyzed for mercury.

Table 6.15. Scientific and common names of fish species captured in EFPC during TVA (1985) and BMAP (Loar 1992; Hinzman 1992) contaminant studies

Common Name	Scientific Name
Bluegill	*Lepomis macrochirus*
Green sunfish	*L. cyanellus*
Redbreast sunfish	*L. auritus*
Warmouth	*L. gulosus*
Largemouth bass	*Micropterus salmoides*
Rock bass	*Ambloplites rupestris*
Common carp	*Cyprinus carpio*
Black redhorse	*Moxostoma duquesnei*
White sucker	*Catostomus commersoni*
Gizzard shad	*Dorosoma cepedianum*

Mean total mercury concentration in bluegill was greatest in samples collected from EFK 22.8 (nearest the Y-12 Plant) at a concentration of 2.13 ± 0.61 mg/kg wet weight (Van Winkle et al. 1984). Normalized mean mercury concentrations in bluegill (adjusted to concentrations expected in a 63-g fish) collected downstream from the Y-12 Plant steadily decreased with increased distance from the Y-12 Plant (1.66, 1.45, and 0.66 mg/kg at EFK 22.7, EFK 13.4, and EFK 2.1, respectively). All of the bluegill collected from EFK 2.1 contained mercury concentrations in excess of background concentrations for bluegill in the geographical area (0.05 mg/kg), based on previous collections made in Melton Hill Reservoir (Elwood 1977). Van Winkle et al. (1984) and Elwood (1977) observed similar longitudinal patterns of decreasing mercury concentrations in fish and surface sediments downstream from the Y-12 Plant, and concluded that there was a sustained mercury source in the headwaters of EFPC, while contaminated sediments were the probable indirect source of mercury to the fish in EFPC.

1984 instream contaminant study. In May through June 1984, TVA conducted an instream contaminant study to determine contaminant concentrations in fish from selected sampling sites, including sites in EFPC (TVA 1985).

To evaluate effects on the fisheries resource, the results are discussed in terms of comparison against U.S. Food and Drug Administration (FDA) action levels. Comparison of these historical data against toxicological benchmarks for protection of biota is included in Sect. 6.4. In EFPC, two to nine fish species, snapping turtles, frogs, and crayfish were collected by electrofishing from three sites [EFK 22.2, EFK 14.2 and EFK 6.4 (Table 6.16)]. Mercury concentrations were highest in fish and other aquatic organisms collected from EFK 22.2. Mean total mercury concentrations in largemouth bass, redbreast sunfish, and frogs (including bullfrogs, green frogs, and pickerel frogs) exceeded 1.0 mg mercury/kg wet weight. In contrast, mean total mercury concentrations in snapping turtles and crayfish were less than 1.0 mg mercury/kg, although both taxa contained individuals having concentrations in excess of 1.0 mg mercury/kg. Even though mean mercury concentrations in redbreast sunfish decreased downstream from the Y-12 Plant, individuals of other fish species having mercury concentrations above 1.0 mg/kg were captured at each site in EFPC.

Organic pollutants were rarely observed in fish from EFPC during the TVA instream contaminant study (TVA 1985). For example, only three such compounds other than PCBs were detected in two fish from one site in EFPC (EFK 22.2). Concentration of bis(2-ethylhexyl) phthalate was at 1.20 mg/kg in a largemouth bass, and aldrin and 4,4-DDE concentrations were at 0.02 and 0.04 mg/kg, respectively, in a carp (*Cyprinus carpio*). PCBs were detected in only three fish species—largemouth bass and carp from EFK 22.2 and a channel catfish (*Ictalurus*

Table 6.16. Total mercury in fish and other aquatic organisms from EFPC, May 1984[a]

Species	Sample location		
	EFK 22.2	EFK 14.2	EFK 6.4
Common carp (*Cyprinus carpio*)	0.77 (0.210-1.3)	NC[b]	NC
Largemouth bass (*Micropterus salmoides*)	1.4 (0.80-1.5)	NC	NC
Bluegill (composite) (*Lepomis macrochirus*)	0.68 (0.54-0.82)	0.80 (0.51-1.0)	0.89 (0.20-1.2)
Redbreast sunfish (*L. auritus*)	1.7 (0.24-3.3)	0.96 (0.64-1.4)	0.67 (0.64-0.70)
Green sunfish (*L. cyanellus*)	NC	NC	0.52[c] (0.52)
Warmouth (*L. gulosus*)	NC	NC	0.96[c] (0.96)
Rock bass (*Ambloplites rupestris*)	NC	NC	1.0[c] (1.0)
Yellow perch (*Perca flavescens*)	NC	NC	0.93)[c] (0.93)
White sucker (*Catostomus commersoni*)	NC	NC	0.97 (0.54-1.4)
Black redhorse (*Moxostoma duquesnei*)	NC	NC	0.57[c] (0.57)
Gizzard shad (composite) (*Dorosoma cepedianum*)	NC	NC	0.12[c] (0.12)
Snapping turtle (*Chelydra serpentina*)	0.72 (0.43-1.2)	0.63 (0.16-1.0)	0.95 (0.41-1.4)
Crayfish	0.82 (0.43-1.2)	0.62 (0.24-0.72)	0.29[c] (0.29)
Frogs	1.6 (U-3.0)[d]	NC	NC

[a]Mercury is measured in mg/kg. Values are mean; ranges are in parentheses.
[b]NC = species was not collected.
[c]Sample size of one individual.
[d]U = below minimum detection amount of 0.10 mg/kg.
Source: Adapted with permission from TVA (1985).

punctatus) from EFK 6.4. Mean total PCB concentrations for the largemouth bass and carp at EFK 22.2 were 0.5 and 2.9 mg/kg, respectively, whereas the channel catfish at EFK 6.4 contained 0.6 mg PCB/kg. Only the carp at EFK 22.2 exceeded the FDA action level of 2.0 mg PCB/kg.

1986–1988 bioaccumulation monitoring programs. During May 1985, bioaccumulation monitoring programs for biota in EFPC were initiated as part of the Y-12 Plant BMAP study (Loar 1992). The first annual report presented data on total mercury, PCB, PAH and ^{137}Cs bioaccumulation in fish and invertebrates collected from EFPC between May 1985 and May 1986 (Loar 1992). The second report on the Y-12 Plant BMAP for EFPC presented data on various inorganic and organic contaminants in aquatic biota in EFPC between December 1986 and May 1988 (Hinzman 1992).

Fish were collected for contaminant analysis from five sites in EFPC (EFK 23.4, EFK 18.2, EFK 13.8, EFK 6.3, and EFK 2.1), plus three reference streams (Brushy Fork, Hinds Creek, and Beaver Creek). The sites in EFPC were chosen to coincide approximately with the sample locations used in studies conducted in 1982 (Van Winkle et al. 1984) and 1984 (TVA 1985). Three reference streams were included in the study to obtain more representative estimates of background concentrations of mercury and PCBs in rural streams similar in size to EFPC, and to attempt to identify a single stream in which all three indicator fish species were abundant. Three fish species (redbreast sunfish, bluegill sunfish, and carp) were collected by electrofishing for contaminant analysis. Fish collections took place in May 1985, December 1985, and May 1986. When possible, eight fish of each species were collected at each site, and only skinless fillets were analyzed for mercury and PCB.

Fish collected from EFPC between May 1985 and May 1986 contained greater mercury concentrations than did fish collected from the reference streams in all three sampling periods [Loar 1992 (Table 6.17)]. Mean mercury concentrations in redbreast sunfish from EFPC were 0.67 mg mercury/kg wet weight, compared with 0.08 mg mercury/kg in redbreast sunfish from the reference streams. Mercury levels in bluegill and carp from EFPC averaged 0.61 and 0.71 mg mercury/kg, respectively, and 0.06 and 0.15 mg mercury/kg, respectively, from the reference streams. Mercury concentrations in sunfish from EFPC were highest at the site closest to the Y-12 Plant and decreased with distance downstream from that site. In the reach of stream immediately below New Hope Pond on the Y-12 Plant site, nearly half of the redbreast sunfish large enough to be taken by fishermen exceeded the 1 mg mercury/kg FDA action limit for total mercury. No sunfish exceeding 1 mg mercury/kg were collected at EFK 13.8 or at any site further downstream. In addition, Loar (1992) reported no indication of increased mercury levels

Table 6.17. Total mercury in redbreast sunfish, bluegill, and carp from EFPC, 1985–1986[a]

Species	Sample location				
	EFK 23.4	EFK 18.2	EFK 13.8	EFK 6.3	EFK 2.1
May 1985					
Redbreast sunfish[b] *(Lepomis auritus)*	0.62±0.08 (12)	0.77±0.09 (9)	0.65±0.07 (8)	0.38±0.02 (8)	0.45±0.12 (7)
Bluegill[c] *(L. macrochirus)*	0.83±0.06 (12)	NC[d]	0.37 (1)	0.65±0.05 (2)	0.48±0.11 (8)
Carp[e] *(Cyprinus carpio)*	0.29±0.10 (2)	NC	1.12±0.26 (5)	0.80±0.05 (8)	0.61±0.02 (2)
December 1985					
Redbreast sunfish *(L. auritus)*	1.26±0.12 (8)	0.83±0.09 (8)	0.73±0.06 (8)	0.56±0.05 (8)	0.36±0.06 (8)
Bluegill *(L. macrochirus)*	0.86±0.08 (8)	0.96 (1)	NC	0.40±0.25 (2)	0.38±0.10 (8)
Carp *(C. carpio)*	NC	NC	0.66±0.07 (8)	0.89±0.09 (8)	NC
May 1986					
Redbreast sunfish *(L. auritus)*	1.19±0.16 (8)	0.87±0.09 (8)	0.59±0.04 (9)	0.41±0.03 (8)	0.34±0.03 (8)
Bluegill *(L. macrochirus)*	0.74±0.12 (9)	0.40 (1)	0.47 (1)	NC	0.29±0.06 (8)
Carp *(C. carpio)*	NC	0.34±0.04 (3)	0.54±0.07 (7)	0.80±0.10 (8)	0.54±0.07 (8)

[a]Mercury is measured in ppm wet weight. Sample size is in parentheses; values are mean ±1 standard error (SE).
[b]Background level (reference stream fish) 0.08 ±0.01 ppm (n=24).
[c]Background level 0.06 ±0.02 ppm (n=12).
[d]NC = no fish collected.
[e]Background level 0.15 ±0.02 ppm (n=16).
Source: Adapted with permission from Loar (1992).

in fish in the vicinity of highly contaminated floodplain areas (upstream of site EFK 13.8) or in fish below the Oak Ridge Sewage Treatment Plant (EFK 13.4), and concluded that these areas are probably not major sources of biologically available mercury to fish. No such trend was observed in carp—fish from the locations further downstream generally had higher mercury concentrations than those collected from the upper reaches of the stream, and individual carp collected from the middle and lower reaches of EFPC exceeded 1 mg mercury/kg.

PCBs were detected in all three fish species during May 1985 through May 1986 [Loar 1992 (Table 6.18)]. Mean PCB concentrations in redbreast and bluegill sunfish (averaged among all five EFPC locations and the three sampling periods) were 0.33 and 0.27 mg PCB/kg, respectively, whereas carp contained a mean concentration of 1.99 mg PCB/kg. Both species of sunfish from the Hinds Creek and Beaver Creek reference sites had mean concentrations of 0.05 mg PCB/kg, whereas carp from Hinds Creek contained 0.07 mg PCB/kg. All sunfish collected from EFPC contained less than 2 ppm. In May 1986, no carp collected from EFPC exceeded 2 ppm total PCBs, in contrast to the May and December 1985 samples of carp, in which 41% and 44% of the carp from EFPC, respectively, exceeded 2 ppm total PCBs.

Contaminant bioaccumulation studies in fish and other aquatic biota continued during December 1986 through May 1988 (Hinzman 1992). Concentrations of mercury in fish from EFPC during this sampling period were elevated above those found in fish from the reference streams (Table 6.19), and were consistent with findings from previous studies (Van Winkle et al. 1984; TVA 1985; Loar 1992). Mean mercury concentrations in redbreast sunfish from EFPC during December 1986 through May 1988 were at 0.92 mg/kg fresh weight, in contrast to 0.08 mg/kg in fish from the reference streams. Bluegill and carp contained mean mercury concentrations of 0.74 and 0.73 mg/kg, respectively, compared with concentrations of 0.08 and 0.21 mg/kg in fish from the reference streams. Thirty percent of all the fish collected from EFPC during this time, including 41% of all redbreast sunfish, exceeded 1 mg mercury/kg. The proportion of fish exceeding 1 mg mercury/kg was higher from 1987 through 1988 than from 1985 through 1986 at all sites except EFK 2.1.

In general, redbreast sunfish at EFK 23.4 had significantly greater mean mercury concentrations than bluegill sunfish at the same site, during both the May 1985 through May 1986 and the December 1986 through May 1988 monitoring periods. However, at EFK 2.1, there were no significant differences in mercury concentrations between the two species.

The downstream decrease in mercury concentration in sunfish observed during May 1985 through May 1986 (Table 6.17) was also observed during December 1986 through May 1988

Table 6.18. Total PCBs in redbreast sunfish, bluegill, and carp from EFPC, 1985–1986[a]

Species	Sample location				
	EFK 23.4	EFK 18.2	EFK 13.8	EFK 6.3	EFK 2.1
May 1985					
Redbreast sunfish[b] *(Lepomis auritus)*	0.48±0.09 (12)	0.34±0.04 (8)	0.39±0.09 (8)	0.25±0.03 (8)	0.19±0.01 (7)
Bluegill[c] *(L. macrochirus)*	0.26±0.04 (8)	NC[d]	NC	0.20±0.16 (2)	0.31±0.05 (8)
Carp[e] *(Cyprinus carpio)*	2.52±2.08 (2)	NC	2.94±0.81 (5)	1.70±0.42 (8)	3.25±2.05 (2)
December 1985					
Redbreast sunfish *(L. auritus)*	0.49±0.09 (8)	0.27±0.08 (8)	0.22±0.02 (8)	0.21±0.02 (8)	0.26±0.05 (8)
Bluegill *(L. macrochirus)*	0.36±0.06 (8)	0.75 (1)	NC	0.24±0.01 (2)	0.20±0.00 (8)
Carp *(C. carpio)*	NC	NC	1.95±0.38 (8)	1.29±0.19 (8)	NC
May 1986					
Redbreast sunfish *(L. auritus)*	0.69±0.15 (9)	0.21±0.04 (8)	0.26±0.05 (8)	0.16±0.04 (8)	0.22±0.06 (8)
Bluegill *(L. macrochirus)*	0.34±0.10 (8)	0.07 (1)	0.16 (1)	NC	0.18±0.04 (8)
Carp *(C. carpio)*	NC	0.83±0.03 (3)	0.75±0.14 (7)	0.44±0.06 (8)	0.44±0.06 (8)

[a]PCBs are measured in ppm wet weight. Sample size is in parentheses; values are mean ± 1 SE.
[b]Background level (reference stream fish) 0.048 ±0.01 ppm (n=15).
[c]Background level 0.06 ±0.01 ppm (n=10).
[d]NC = no fish collected.
[e]Background level 0.07 ±0.01 ppm (n=7), excluding two fish containing 3.2 and 0.78 ppm PCBs that were collected from the reference stream (Hinds Creek). PCB analyses of sediments and sunfish from this stream did not indicate the presence of contamination. The source of PCBs found in these two fish is not known; however, they do not appear to have acquired such PCB levels at the site where they were collected.
Source: Adapted with permission from Loar (1992).

Table 6.19. Total mercury in redbreast sunfish, bluegill, and carp from EFPC, 1986–1988[a]

Species	Sample location					
	EFK 23.4	EFK 18.2	EFK 13.8	EFK 10.0	EFK 6.3	EFK 2.1
December 1986						
Redbreast sunfish[b] *(Lepomis auritus)*	1.70±0.10 (8)	0.91±0.05 (8)	0.91±0.06 (8)		0.70±0.05 (8)	0.40±0.03 (8)
Bluegill[c] *(L. macrochirus)*	1.16±0.16 (7)	NC[d]	0.31 (1)		0.55 (1)	0.38±0.08 (8)
Carp[e] *(Cyprinus carpio)*	NC	NC	NC	1.04±0.16 (5)	0.83±0.05 (8)	NC
May 1987						
Redbreast sunfish *(L. auritus)*	1.49±0.13 (8)	1.15±0.08 (8)	0.83±0.06 (8)		0.82±0.04 (8)	0.47±0.09 (8)
Bluegill *(L. macrochirus)*	0.70±0.11 (8)	NC	NC		0.60±0.14 (3)	0.38±0.08 (9)
Carp *(C. carpio)*	0.11 (1)	0.31 (1)	0.51±0.09 (6)	NC	0.66±0.13 (8)	0.66±0.02 (6)
December 1987						
Redbreast sunfish *(L. auritus)*	1.57±0.58 (8)	1.25±0.11 (8)	1.03±0.08 (8)		0.75±0.08 (8)	0.43±0.04 (8)
Bluegill *(L. macrochirus)*	1.76±0.28 (8)	NC	NC		0.42 (1)	0.43±0.06 (8)
Carp *(C. carpio)*	NC	0.83±0.08 (8)	NC	NC	0.82±0.09 (8)	0.56 (1)
May 1988						
Redbreast sunfish *(L. auritus)*	1.45±0.21 (8)	0.95±0.16 (8)	0.66±0.08 (8)		0.60±0.06 (8)	0.35±0.03 (8)
Bluegill *(L. macrochirus)*	1.08±0.28 (8)	NC	NC		NC	0.32±0.03 (8)
Carp *(C. carpio)*	NC	0.82±0.13 (8)	0.99±0.16 (8)	NC	0.62±0.13 (6)	0.53±0.09 (8)

[a]Mercury is measured in μg/g wet weight. Sample size is in parentheses; values are mean ±SE.
[b]Background level (reference stream fish) 0.08 ±0.0 μg/g (n=30).
[c]Background level 0.08 ±0.01 μg/g (n=25).
[d]NC = no fish collected.
[e]Background level 0.21 ±0.03 μg/g (n=9).
Source: Adapted with permission from Hinzman (1992).

(Table 6.19). Mercury concentrations in both species were highest at EFK 23.4 (exceeding 1 mg mercury/kg, except for bluegill during the May 1987 sampling) and steadily decreased to a range of 0.32 to 0.47 mg/kg at EFK 2.1 (Table 6.19). Mercury concentrations in carp generally did not decrease with increased downstream distance from the Y-12 Plant, but the scarcity of data for carp from EFK 23.4 and EFK 18.2 (Table 6.19) makes it difficult to assess the mercury bioaccumulation in carp at these two sites.

Mean concentrations of mercury in redbreast sunfish collected from all the BMAP sampling sites in EFPC (except EFK 2.1) were significantly greater (linear regression of mercury concentration in fish versus time, P level not stated) during the December 1986 through May 1988 sampling than during the May 1985 through May 1986 sampling (Hinzman 1992). A significant temporal increase in mercury concentration in bluegill also was observed at EFK 23.4 during May 1985 through May 1988, but no significant change was observed at EFK 2.1 (Hinzman 1992). A significant temporal increase in mercury concentration in carp was observed only at EFK 18.2. Hinzman concluded that mercury concentrations in fish from EFPC exhibited substantial variability through time, and that the mercury levels in fish collected from December 1986 through May 1988 were within the ranges typically observed during previous investigations in 1982 (Van Winkle et al. 1984) and 1984 (TVA 1985).

According to Hinzman (1992), a study was conducted to assess the availability of sediment-associated mercury to the blacknose dace (*Rhinicthys atralatus*), a native fish in EFPC. The fish were maintained in separate 30-L aquaria containing surface sediments (<2 mm, wet sieved) from New Hope Pond or from a site in EFPC 5 km downstream (EFK 18.2). Mercury concentrations in the sediments were 153 and 62 mg/kg dry weight, respectively. Control group fish were maintained in the laboratory in an aquarium supplied with continuous renewal of fresh, dechlorinated process water. Hinzman concluded that mercury concentrations in the dace from all three treatments were the same after 25 weeks of exposure.

PCB contamination was detected in sunfish and carp from EFPC during the December 1986 through May 1988 Y-12 Plant BMAP collections [Table 6.20 (Hinzman 1992)]. Mean PCB concentration in redbreast and bluegill sunfish (averaged among all five sample sites in EFPC during the four sampling periods) was 0.46 mg/kg, whereas carp contained a mean concentration of 0.98 mg PCB/kg. Mean concentrations of PCBs in redbreast sunfish, bluegill, and carp collected from the Hinds Creek reference stream were 0.04, 0.03, and 0.08 mg/kg, respectively. Maximum concentrations of PCBs in the three species were 3.8 mg/kg in redbreast sunfish, 2.2 mg/kg in bluegill, and 3.2 mg/kg in carp. Only 5.6% of all sunfish collected from EFPC during the sampling period contained PCB concentrations in excess of 2 mg PCB/kg, and all

Table 6.20. Total PCBs in redbreast sunfish, bluegill, and carp from EFPC, 1986–1988[a]

Species	Sample location					
	EFK 23.4	EFK 18.2	EFK 13.8	EFK 10.0	EFK 6.3	EFK 2.1
December 1986						
Redbreast sunfish[b] *(Lepomis auritus)*	1.74±0.37 (8)	0.28±0.06 (8)	0.31±0.06 (8)		0.10±0.01 (8)	0.08±0.01 (8)
Bluegill[c] *(L. macrochirus)*	0.70±0.15 (7)	NC[d]	NC		0.05 (1)	0.12±0.03 (8)
Carp[e] *(Cyprinus carpio)*	NC	NC	NC	0.52±0.08 (5)	1.22±0.19 (5)	NC
May 1987						
Redbreast sunfish *(L. auritus)*	1.04±0.40 (8)	0.57±0.11 (8)	0.30±0.07 (8)		0.13±0.04 (8)	0.15±0.04
Bluegill *(L. macrochirus)*	0.46±0.11 (8)	NC	NC		0.13±0.02 (3)	0.17±0.06 (9)
Carp *(C. carpio)*	0.59 (1)	0.97 (1)	0.96±0.18 (6)	NC	0.82±0.24 (8)	1.11±0.21 (6)
December 1987						
Redbreast sunfish *(L. auritus)*	0.61±0.27 (8)	0.76±0.17 (8)	0.62±0.15 (8)		0.18±0.03 (8)	0.14±0.03 (8)
Bluegill *(L. macrochirus)*	1.45±0.21 (8)	NC	NC		0.31 (1)	0.14±0.04 (8)
Carp *(C. carpio)*	NC	2.03±0.36 (8)	NC	NC	0.62±0.08 (8)	0.17 (1)
May 1988						
Redbreast sunfish *(L. auritus)*	0.86±0.18 (8)	0.43±0.07 (8)	0.36±0.06 (8)		0.19±0.03 (8)	0.18±0.02 (8)
Bluegill *(L. macrochirus)*	0.66±0.23 (8)	NC	NC		NC	0.27±0.05 (8)
Carp *(C. carpio)*	NC	1.11±0.16 (8)	1.52±0.28 (8)	NC	0.51±0.12 (6)	0.28±0.06 (8)

[a]PCBs are measured in μg/g wet weight. Sample size is in parentheses; values are mean ±SE.
[b]Background level (reference stream fish) 0.04 ±0.01 μg/g (n=31).
[c]Background level 0.03 ±0.01 μg/g (n=31).
[d]NC = no fish collected.
[e]Background level 0.18 ±0.05 μg/g (n=9).
Source: Adapted with permission from Hinzman (1992).

sunfish that exceeded 2 mg PCB/kg were collected from EFK 23.4 (Hinzman 1992). In contrast, no sunfish collected at EFK 23.4 during the May 1985 through May 1986 collections exceeded 2 mg PCB/kg (Loar 1992). As compared with sunfish, a greater proportion of carp (11%) collected from EFPC during the December 1986 through May 1988 sampling exceeded 2 mg PCB/kg, and these were collected from all sites except EFK 2.1 and EFK 23.4 (where carp are rarely found). Mean concentrations of PCBs in redbreast sunfish and bluegill were not significantly different where both species were abundant (T-test, P level not stated). However, mean PCB concentrations in carp generally were three- to five-fold greater than in sunfish at sites where both were collected, and were significantly greater at all sites in EFPC when the sampling periods were pooled (except EFK 23.4, where no statistical analysis was performed because of the small sample size).

The longitudinal pattern of decreasing PCB concentrations in sunfish as a function of downstream distance from the Y-12 Plant, observed during the May 1985 through May 1986 collections (Table 6.18), was also observed during the December 1986 through May 1988 sampling [Table 6.20 (Loar 1992)]. The relationship between PCB concentrations in redbreast sunfish and distance downstream from New Hope Pond was statistically significant (T-test, P level not specified) in all sampling periods. Bluegill were collected in adequate numbers for analysis at EFK 23.4 and EFK 2.1, and the decrease in PCB concentrations between the two sites was statistically significant on all sampling periods except May 1988. In carp, the relationship between PCB concentration and downstream distance from New Hope Pond was significant during the December 1987 and May 1988 collections, but not during the two previous sampling periods. Hinzman (1992) concluded that the consistent pattern of gradual downstream decrease in PCB concentrations in sunfish in EFPC "indicated the presence of a continuing input of biologically available PCBs to EFPC upstream from EFK 23.4" (p. 4-29). However, a specific source was not identified.

Substantial changes in mean PCB concentrations in sunfish were commonly observed between successive sampling periods at specific sites. Concentrations of PCBs in fish in EFPC did not appear to follow any consistent increasing or decreasing trend during May 1985 through May 1988 (Hinzman 1992). Hinzman concluded that the high degree of variation among sampling periods indicated that PCB concentrations in sunfish in EFPC can fluctuate fairly rapidly.

Two studies were conducted during April through May 1987 and 1988 in which Asiatic clams (*Corbicula fluminea*) in polypropylene cages were placed in various locations upstream and downstream from the New Hope Pond outfall for four weeks in an attempt to locate the source

of PCB contamination in EFPC [Table 6.21 (Hinzman 1992)]. Mean PCB concentrations in the clams from three downstream sites ranged from 0.40 to 0.57 mg/kg wet weight in the 1987 study, and from 0.32 to 0.50 mg/kg during the 1988 study. The downstream pattern of PCB accumulation in clams did not show the same consistent decrease with distance from the Y-12 Plant that was observed in fish (Hinzman 1992). Hinzman was unable to provide an explanation for the dissimilar trends but did conclude that clam data from EFPC sites near the Y-12 Plant may underestimate the bioavailability of PCBs compared with sites further downstream. Conditions at EFK 23.4 had been shown to produce toxic effects in native clams (*Sphaerium fabale*) that were transplanted to the site.

Asiatic clams from Bull Run in Union County, Tennessee, were placed in cages at EFK 23.4 for 4 weeks during May and June of 1988 to determine the bioaccumulation of PAHs (Hinzman 1992). A portion of the clams were saved and analyzed for 14 PAHs to serve as a control. Mean concentrations of benzo(a)anthracene, benzo(b)fluoranthene, chrysene, and pyrene in clams from EFK 23.4 were more than four-fold greater than the concentrations in the control group of clams (no statistical test or P value was stated) (Table 6.22). No individual PAH concentration in the clams from EFK 23.4 exceeded 1.0 mg/kg. Of the 14 PAHs detected in the clams, benzo(b)fluoranthene had the greatest concentration (0.35 mg/kg). Hinzman concluded that the pattern of PAH accumulation in the clams from EFPC was indicative of the presence of petroleum or coal-derived hydrocarbon mixtures in water. These mixtures would contain a large suite of PAHs, and the more water-soluble compounds such as pyrene and benzo(a)anthracene would be likely to accumulate in aquatic biota.

In February and May 1987, periphyton from three rocks at each of five sites in EFPC and at one reference site in Brushy Fork Creek was analyzed for inorganic contaminants (Hinzman 1992). The EFPC sample locations were at EFK 23.2, EFK 17.0, EFK 13.8, EFK 10.6, and EFK 6.3; the Brushy Fork reference site location was at Brushy Fork Kilometer 7.6. Cadmium, copper, lead, nickel, silver, and zinc concentrations in periphyton were greater at EFK 23.2 than at the rest of the EFPC sites or the Brushy Fork reference site during both sampling periods (Table 6.23). Mercury concentrations in the periphyton were measured only during the February 1987 sampling. Periphyton mercury concentrations in EFPC decreased with downstream distance from the Y-12 Plant (from levels of 159 mg/kg dry weight at EFK 23.2 to 16 mg/kg dry weight at EFK 6.3). All sites exceeded the periphyton mercury concentration of 0.3 mg/kg dry weight found at the Brushy Fork reference site (Table 6.23).

1990 small mammal study. Small mammals were trapped in the EFPC watershed area during 1986 and 1987 as part of a larger three-year biomonitoring study of several sites on ORR

Table 6.21. PCB concentrations in Asiatic clams (*Corbicula fluminea*) held in cages in EFPC downstream from New Hope Pond, April 23–May 26, 1987, and April 8–May 6, 1988[a]

Year	Sample location				
	EFK 23.4	EFK 18.2	EFK 13.8	EFK 6.3	Reference
1987	0.57 ± 0.20	0.50 ± 0.06	0.49 ± 0.05	—	0.08 ± 0.02
1988	0.32 ± 0.04	—	0.50 ± 0.06	0.33 ± 0.02	0.05 ± 0.01

[a]PCB values are measured in μg/g wet weight. Values are mean ± standard deviation; n=3 composites containing ~ 10 clams each.
[b]Reference site for clams was Beaver Creek in 1987, Bull Run in 1988.
Source: Adapted with permission from Hinzman (1992).

Table 6.22. PAH concentrations in Asiatic clams *(Corbicula fluminea)* before and after being transplanted to EFPC reference site, May 18–June 17, 1988[a]

PAH	Sample location	
	EFK 23.4	Bull Run
Acenaphthene	0.14 ± 0.06	<0.06
Anthracene	<0.05	<0.05
Benzo(a)anthracene	0.037 ± 0.010	<0.001
Benzo(a)pyrene	0.070 ± 0.012	0.035 ± 0.005
Benzo(b)fluoranthene	0.35 ± 0.07	<0.06
Benzo(g,h,i)perylene	0.088 ± 0.002	0.025 ± 0.005
Benzo(k)fluoranthene	0.13 ± 0.10	0.04 ± 0.00
Chrysene	0.15 ± 0.01	<0.01
Dibenz(a,h)anthracene	0.010 ± 0.000	0.009 ± 0.000
Fluoranthene	<0.50	<0.50
Napthalene	0.31 ± 0.10	<0.20
Phenanthrene	0.065 ± 0.013	<0.020
Pyrene	0.15 ± 0.02	0.035 ± 0.005
Indenopyrene	0.25 ± 0.03	0.12 ± 0.00

[a]PAHs are measured in μg/g wet weight. Values are mean ± standard error, n=4 for EFK 23.4, n=2 for Bull Run. Samples are composites of ~ 10 clams each.
Source: Adapted with permission from Hinzman (1992).

Table 6.23. Concentrations of selected metals in periphyton, February and May 1987[a]

Sampling dates	Sample location					
	EFK 23.2	EFK 17.0	EFK 13.8	EFK 10.6	EFK 6.3	Reference[b]
Ag						
Feb 1987	21	<10	<9.9	<10	<10	<10
May 1987	<32	< 7	<5	< 9	< 7	7
Cd						
Feb 1987	31	6.9	4.6	4.2	2.4	1.3
May 1987	39.7	5.1	4	4.5	2	1
Cu						
Feb 1987	207	68	59	45	29	15
May 1987	137	50	42	40	33	11
Hg						
Feb 1987	159±44	56±3.5	23±13	14±3.2	16±6.2	0.3±0.06
May 1987	NA	NA	NA	NA	NA	NA
Ni						
Feb 1987	233	51	31	50	26	22
May 1987	580	35	43	74	23	24
Pb						
Feb 1987	63	66	<40	<40	<39	<40
May 1987	<128	54	36	36	<26	<28
Zn						
Feb 1987	3500	633	433	500	217	557
May 1987	3800	360	233	410	207	437

[a]Metals are measured in ppm dry weight. Values are means from three replicates.
[b]Reference site was in Brushy Fork (BFK 7.6).
Source: Adapted with permission from Hinzman (1992).

(Talmage and Walton 1990). A total of 34 animals representing three species were found in traps and their kidneys were analyzed for mercury concentrations. The mean mercury concentration in white-footed mouse (*Peromyscus leucopus*) kidney was 4.2 μg/g dry weight. This level was 2.3 times higher than that at the reference site and was significant ($p < 0.01$). The mean mercury level in shorttail shrew (*Blarina brevicauda*) was 137.0 μg/g dry weight, 42 times higher than that measured in animals at the reference site. These elevated levels were also significantly higher than that at the reference site ($p < 0.005$). Only one individual of cotton rat (*Sigmodon hispidus*) was trapped at the EFPC site with a reported mercury level of 6.7 μg/g dry weight. The elevated mercury levels in the mammals trapped in this study correlated positively with elevated levels of mercury in the site soil.

An additional, unpublished study (Talmage 1991) reported elevated levels of mercury in white-footed mouse and shorttail shrew (1.16 and 38.8 ppm mercury wet weight, respectively) as compared to the same species at a reference site. This study also reported levels of mercury in earthworms higher than those at the reference site.

1982 Mercury concentration study - terrestrial portion. Liverwort (*Marchantia* sp.) and moss (species not identified) were collected generally 10 to 15 cm (4 to 6 in.) above the water line at EFK 22.2, EFK 7.7, and EFK 2.1 (Van Winkle et al. 1984). Pasture grass was harvested along transects on the floodplain of EFPC at EFK 13.4 and EFK 8.8 and at a control area near the X-10 Site. Clip plots were located at 5, 30, and 100 m (330 ft) from the stream bank, and three replicate samples each of standing live foliage (i.e., of the 1982 growing season, having experienced no flooding events) and dead foliage (i.e., of the 1981 growing season, having been subjected to several flooding events) were collected at each distance.

Mean concentrations of mercury in moss and liverwort at EFK 22.2 were similar (34.0 and 35.7 mg mercury/kg dry weight, respectively) and were more than double the mercury concentrations in moss at EFK 7.7 and EFK 2.1 (14.0 and 14.3 mg mercury/kg dry weight, respectively).

In the pasture grass study of Van Winkle et al. (1984), mercury concentrations in live and dead foliage from the nonflooded control area at the X-10 Site were similar (0.10 and 0.12 mg mercury/kg wet weight, respectively). Van Winkle et al. thus concluded that mercury uptake from soil is not a significant pathway for mercury incorporation into plants. Furthermore, the authors concluded that atmospheric deposition of mercury was not significant, because dead foliage was exposed to atmospheric deposition for a much longer period than was live foliage but contained approximately the same concentration of mercury. Mercury concentrations in dead

foliage collected at 30 and 5 m (99 and 16.5 ft) from the creek edge at EFK 13.4 were 2.1 and 4.4 mg/kg, respectively, 21- and 44-fold greater than mercury concentrations in vegetation at control sites. In contrast, live foliage contained only 0.23 and 0.18 mg mercury/kg at 5 and 30 m (16 and 99 ft), respectively. At EFK 8.8, dead foliage collected 5 m (16 ft) from the edge of the creek contained 6.97 mg mercury/kg (nearly 60-fold greater than for the control sample), whereas dead foliage 30 m (99 ft) from the creek edge contained only 0.42 mg mercury/kg (Van Winkle et al. 1984). Mercury concentrations in live foliage at 5 and 30 m (16 and 99 ft) from the creek edge (0.11 and < 0.10 mg mercury/kg, respectively) were nearly identical to the mercury concentrations in the control sample (0.10 mg mercury/kg). These results suggest that the elevated concentrations of mercury on the grasses were primarily a result of surface deposition of sediments.

1987 vegetation uptake study. A study of native vegetation was conducted by ORAU (Gist 1987) to measure the transfer of contaminants from soils to plants in the EFPC floodplain. Johnson grass (*Sorghum halepense*), honeysuckle (*Lonicera japonica*), sneeze weed (*Helenium* sp.) and jewel weed (*Impatiens bilora*) from the floodplain were sampled and partitioned into roots, stems, leaves, and fruit portions, which were then individually analyzed for mercury concentrations. There appeared to be little or no mercury uptake into any portion of these plants growing in the high mercury soils.

This study also reported mercury concentrations in two white-tailed deer (*Odocoileus virginianus*), killed in vehicle/deer collisions on a portion of the Oak Ridge Turnpike paralleling the floodplain, to be 0.007 ppm mercury in muscle and 0.01 ppm in liver. These levels do not indicate that there is significant accumulation of mercury in these deer from the vegetation in the EFPC floodplain.

6.2.3.2 Current exposure profile

The measured concentrations of mercury in redbreast sunfish, stonerollers, benthic macroinvertebrates, crayfish, earthworms (including gut contents), and flying insects with aquatic juveniles (5 sites) and with terrestrial juveniles (2 sites) were elevated over those at the reference site. Concentrations in fish, crayfish, and earthworms generally decreased with increasing distance from the Y-12 Plant. Uranium concentrations in stonerollers at the four EFPC sites closest to the Y-12 Plant, benthic macroinvertebrates, earthworms, and flying insects with terrestrial juveniles were elevated over those at the reference site. The concentration of Aroclor 1260, the major PCB contaminant in EFPC, in fish, crayfish, and flying insects was also above concentrations in organisms from the reference stream and decreased with increasing

distance downstream from the Y-12 Plant. Various other inorganics, pesticides, and PCBs were also elevated in various indicator organisms at different EFPC sites. Summaries of analytical results on whole-body concentrations of 52 contaminants in composite samples of redbreast sunfish, stonerollers, benthic macroinvertebrates, and crayfish collected from EFPC and Hinds Creek are presented in Appendix Q, Table Q.2. The list of contaminants includes mercury, uranium and 6 other inorganic elements, 7 PCB mixtures, 21 pesticides, and 16 PAH compounds. Tables 6.24 for aquatic indicator organisms and 6.25 for terrestrial indicator organisms summarizes the major trends observed for whole-body concentrations of mercury, PCBs, PAHs, pesticides, and other inorganic contaminants. Discussions about specific concentrations of contaminants in aquatic and terrestrial biota are presented in the following subsections, arranged by taxa and contaminant.

Fish

Sampling methods and population survey results for fish are presented and discussed in Sect. 6.3.3.1.

Mercury. Whole-body mercury concentrations in redbreast sunfish and stonerollers collected from each of the six sample sites in EFPC ranged from 1.8 to 0.2 mg/kg and from 6.4 to 0.4 mg/kg, respectively, but were only 0.08 and 0.4 mg/kg, respectively, in these two species collected from Hinds Creek (Fig 6.42). Whole-body mercury concentrations in both redbreast sunfish and stonerollers collected from the three sites closest to the Y-12 Plant [sites 1, 2, and 3 (Fig. 6.42)] all exceeded the FDA action level of 1.0 mg mercury for fish sold for human consumption (49 *FR* 45663). Maximum whole-body mercury concentrations of 4.9 mg/kg in redbreast sunfish and 6.4 mg/kg in stonerollers were obtained at Sites 3 and 2, respectively. Mercury concentrations in redbreast sunfish decreased steadily downstream from 1.8 mg/kg at Site 3 to 0.2 mg/kg at Site 6, and decreased in stonerollers from 6.4 mg/kg at Site 2 to 0.4 mg/kg at Site 6. Whole-body mercury concentrations in redbreast sunfish from all sample sites in EFPC exceeded those from the reference site, whereas only stonerollers from Site 6 had whole-body mercury levels less than those from the reference site.

Linear correlation coefficients between mercury concentrations in surface water and mercury body burdens in fish were not significant, regardless of whether the regression analysis included all sites in EFPC, or just the three sites closest to the Y-12 Plant. However, the poor statistical correlations were due to the unusually high mercury body burdens at a single site for each fish species (3 for redbreast sunfish; 2 for stonerollers). The unusually high body burdens in fish from these two sites were probably due to a site-specific increased exposure via the

Table 6.24. Summary table of trends for whole-body concentrations of contaminants in aquatic biota collected from EFPC and Hinds Creek, October 7-29, 1991

Taxon	Mercury	Other inorganics	PCBs		Pesticides	PAHs
			Aroclor 1260	Other		
Redbreast Sunfish	• In samples from all six Sites in EFPC and Hinds Creek. • Maximum at Site 3. • Decreasing body burdens downstream from Sites 3 to 6. • All samples in EFPC exceeded concentrations in sample from Hinds Creek.	• Uranium was below sample detection at all Sites.	• In samples from all six Sites in EFPC and Hinds Creek. • Maximum level at Site 3. • Decreasing body burdens downstream from Sites 3 to 6. • All samples in EFPC exceeded concentrations in sample from Hinds Creek.	• All other mixtures were below sample detection limits.	• Most were less than sample detection limit. • Chlordane, dieldrin, and heptachlor were in samples from all Sites including Hinds Creek. • Decreasing body burdens from Sites 1 to 2, then increasing to maximum at Sites 4 or 5. • Nearly all Sites in EFPC exceeded Hinds Creek.	• All individual PAHs were below sample detection limits except for acenaphthene. • Acenaphthene body burden was greatest at Site 1, then nearly constant at remaining EFPC Sites. • All samples in EFPC exceeded concentrations in samples from Hinds Creek.
Stoneroller	• In all six samples from EFPC and Hinds Creek. • Maximum at Site 2. • Decreasing body burdens downstream from Sites 2 to 6. • All samples in EFPC except Site 6 exceeded Hinds Creek sample.	• Uranium in samples from all Sites. • Maximum body burdens at Site 1. • Decreasing body burdens downstream from Sites 1 to 6. • Sites 1 to 4 exceeded Hinds Creek sample.	• Sufficient sample at Sites 1 to 5 and Hinds Creek. • Maximum at Site 1. • Decreasing body burdens downstream from Sites 1 to 4. • All samples in EFPC exceeded concentrations in samples from Hinds Creek.	• All the mixtures were below sample detection limit.	• Same Sites as for PCBs. • Maximum at Site 1. • General trend of decreasing body burdens Sites 1 to 4. • All samples in EFPC exceeded concentrations in samples from Hinds Creeks.	• Same Sites as for PCBs. • Maximum at Site 1. • Same trend as for pesticides. • Nearly all Sites in EFPC exceeded sample from Hinds Creek.

Table 6.24 (continued)

Taxon	Mercury	Other inorganics	PCBs		Pesticides	PAHs
			Aroclor 1260	Other		
Benthic macro-inverte-brates	• Sufficient sample only at 4 Sites in EFPC and Hinds Creek. • Maximum at Site 2; other Sites similar to each other. • All samples from EFPC exceeded sample from Hinds Creek.	• Same Sites as for uranium and mercury. • Same trends as for mercury.	• Sufficient sample only at 2 Sites in EFPC (Sites 1 and 2). • Maximum at Site 1 (6-fold greater than at Site 2).	• All other mixtures below sample detection limit.	• Same Sites as for PCBs. • Nearly all were below sample detection limits.	• Same Sites as for PCBs. • Most individual PAHs were similar to concentrations in samples from the two Sites. • Fluoranthene was maximum at Site 1. • Benzo(a)pyrene was maximum at Site 2.
Crayfish	• Sufficient sample at 4 Sites in EFPC and Hinds Creek. • Maximum at Site 1. • Decreasing body burdens downstream. • All samples from EFPC exceeded concentrations in samples from Hinds Creek.	• Same Sites as for mercury. • Uranium was below sample detection limit at most Sites. • Selenium body burdens were greatest in samples for Site 3, and all EFPC samples exceeded concentrations in samples from Hinds Creek.	• Same Sites as for mercury. • Same trends as for mercury.	• All other mixtures below sample detection limit.	• Same Sites as for mercury. • Nearly all were below sample detection limits.	• Sufficient sample only at Sites 1 to 3, and Hinds Creek. • Maximum at Site 3. • Most individual PAHs increased downstream from Sites 1 to 3. • Most PAHs at the 3 Sites in EFPC exceeded concentrations in sample from Hinds Creek.

Table 6.25 Summary table of trends for whole-body contaminant concentrations in terrestrial biota collected from EFPC and reference site in late 1991

Taxon	Mercury	Other inorganics	PCBs		Pesticides	PAHs
			Aroclor 1260	Other		
Mice	• Levels below detection limit in 9 of 11 individuals. • Maximum value of 1105 μg/kg at Site 2.	• Antimony, chromium, and selenium measured above detection limit in >50% of individuals.	• Above detection limits in all samples except reference. • Range from 51 μg/kg to 480 μg/kg, with highest at Site 1.	• All others below detection limit.	• Nearly all were below detection limits. • One value for DDT and one for heptachlor oxide above detection limits.	• All levels below detection limits or twice reference levels.
Shrews	• Maximum of 7.9 mg/kg at Site 5.	• Uranium not detected. • Antimony, chromium, and selenium similar to mice.	• Maximum of 1400 μg/kg at Site 5.	• Not detected.	• Nearly all were below detection limits.	• Not analyzed.
Vole	• Less than detection limit.	• Chromium levels similar to mice. • Uranium not detected.	• Reported at 46 μg/kg.	• Aroclor 1016 at 73 μg/kg.	• All below detection limit.	• No unqualified levels above unqualified detection limits.

Table 6.25 (continued)

Taxon	Mercury	Other inorganics	PCBs		Pesticides	PAHs
			Aroclor 1260	Other		
Heron	• Breast feathers at 5.3 mg/kg • Liver sample at 9.0 mg/kg.	• Chromium, selenium, and zinc above detection limits in feathers. • Selenium and zinc above detection in liver.	• Liver sample at 1.4 mg/kg.	• All others below detection limit.	• Several reported but most below the detection limit.	• No unqualified levels above detection limit.
Wrens	• Levels at 3.5 mg/kg in both samples.	• Antimony, selenium, and EMC above levels in terrestrial insects.	• Average of 2100 μg/kg is 10 times average in terrestrial insects.	• No other detected.	• Only 4 detected.	• None above detection limit.
Flying insects	• Most samples below detection limit. • Maximum levels of 3189 μg/kg at Site 3. • No correlation between measure concentrations in insects and soils.	• Uranium and cadmium not detected. • Antimony, chromium, zinc, arsenic and selenium noted above detection levels at 2 sites.	• Range of 20-350 μg/kg. • Maximum at Site 2 and decreased steadily with distance from Y-12 Plant.	• All levels below detection or reference site levels.	• DDE reported at Site 3, chlordanes at Site 4. • Others not identified above twice reference site levels.	• Few detected; highest value at Site 3.

Table 6.25 (continued)

Taxon	Mercury	Other inorganics	PCBs		Pesticides	PAHs
			Aroclor 1260	Other		
Earthworms	• Range from 5-33 mg/kg. • Maximum observed at Site 2, with a steady decrease downstream. • No good correlation between earthworm composites and soil levels.	• Arsenic, cadmium, selenium, uranium, zinc, and chromium levels above reference site.	• Not sufficient sample for analysis.	• Not sufficient sample for analysis.	• Not sufficient sample for analysis.	• Not sufficient sample for analysis.
Grass/ vegetables	• Geometric mean grass/browse concentration of 0.34 mg/kg, with maximum of 17 mg/kg. • Grasses near creek showed higher levels. • Levels in vegetables ranged from <0.03 - 3.2 mg/kg.	• Data not available.	• Not measured.	• Not measured.	• Not measured.	• Not measured.

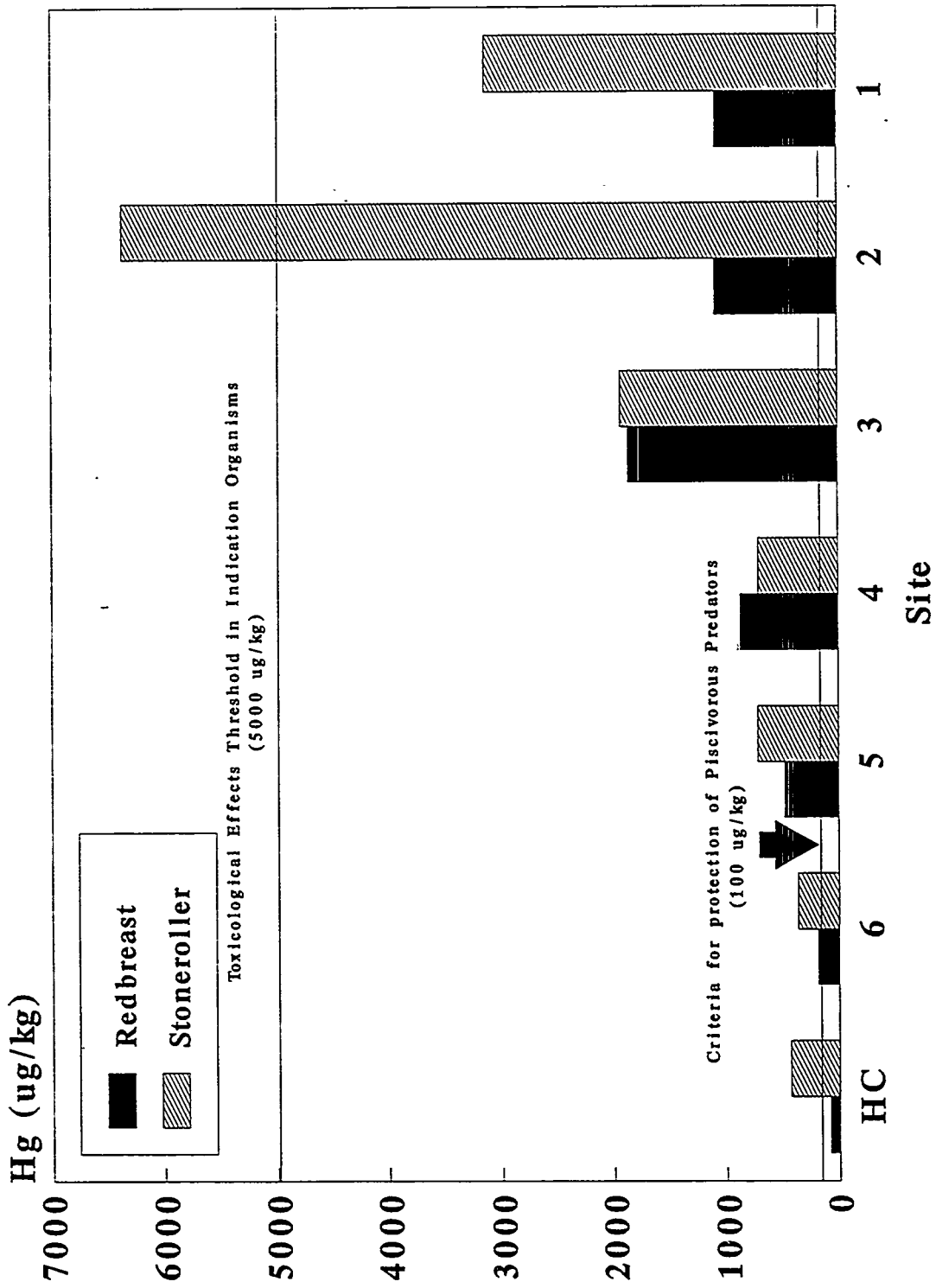

Fig. 6.42. Whole-body mercury concentrations in redbreast sunfish and stonerollers collected from EFPC and Hinds Creek, October 7-12, 1991.

ingestion pathway, which overshadowed the body burdens that would have been observed if the exposure had been limited to the water-borne contaminants alone.

The high mercury body burdens in redbreast sunfish at Site 3 may have been due in part to the presence of terrestrial insects that contained high mercury body burdens (3.19 mg/kg), coupled with site-specific physical environmental factors that would enhance the likelihood that these insects would be consumed by sunfish. The stream flow at Site 3 was mostly one long run, fairly deep, with very slow flow velocity except at the riffle. The slow flow velocity would increase the probability that insects would land on the water surface, where they could be consumed by sunfish. Sunfish were often observed feeding on insects at the water surface at Site 3 during field sampling for the fish, benthic macroinvertebrates, and periphyton studies comprising this ERA. Although the terrestrial insects comprised an unknown proportion of the redbreast sunfish diet, the high body burdens of mercury in the insect composite sample suggest that even if the proportion of terrestrial insects consumed was small, the increased dose of mercury might account for the elevated mercury levels in sunfish.

The increased body burdens of mercury in stonerollers at Site 2 were probably due to an increased dose of mercury to the stonerollers from eating periphyton containing elevated mercury concentrations. Although mercury concentrations in periphyton were not measured during this study, it is reasonable to assume that the mercury concentrations would be elevated in periphyton at Site 2. Mercury bound to fine-particulate sediment or organic matter being discharged from Lake Reality would be more likely to settle out onto stream substrates (including periphyton) in the slower moving stretch of EFPC at Site 2 than at the swifter-flowing stretch at Site 1. Thus, at Site 2, the increased deposition of water-borne fine particulate matter containing enriched concentrations of mercury may have resulted in an additional dose of mercury to stonerollers (Fig. 6.43). The additional dose of mercury could account for the elevated body burdens of this contaminant in stonerollers at this site.

PCBs. In redbreast sunfish and stonerollers, Aroclor 1260 was the only PCB mixture present above the sample detection limit. Whole-body mean Aroclor 1260 concentrations in redbreast sunfish and stonerollers collected from each of the six sample sites in EFPC ranged from 0.47 to 2.8 mg/kg and from 0.82 to 8.1 mg/kg, respectively, but were only 0.007 and 0.003 mg/kg, respectively, in these two species collected from Hinds Creek (Fig. 6.44). Because of insufficient sample mass, no analysis was conducted for stonerollers at Site 6. Aroclor 1260 concentrations in redbreast sunfish decreased steadily downstream from 2.8 mg/kg at Site 3 to 0.47 mg/kg at Site 6, and also decreased in stonerollers from 8.1 mg/kg at Site 1 to 0.82 mg/kg at Site 4. Mean Aroclor 1260 concentrations in both species collected from all sites in EFPC were greater than the concentrations in the fish collected from the reference site.

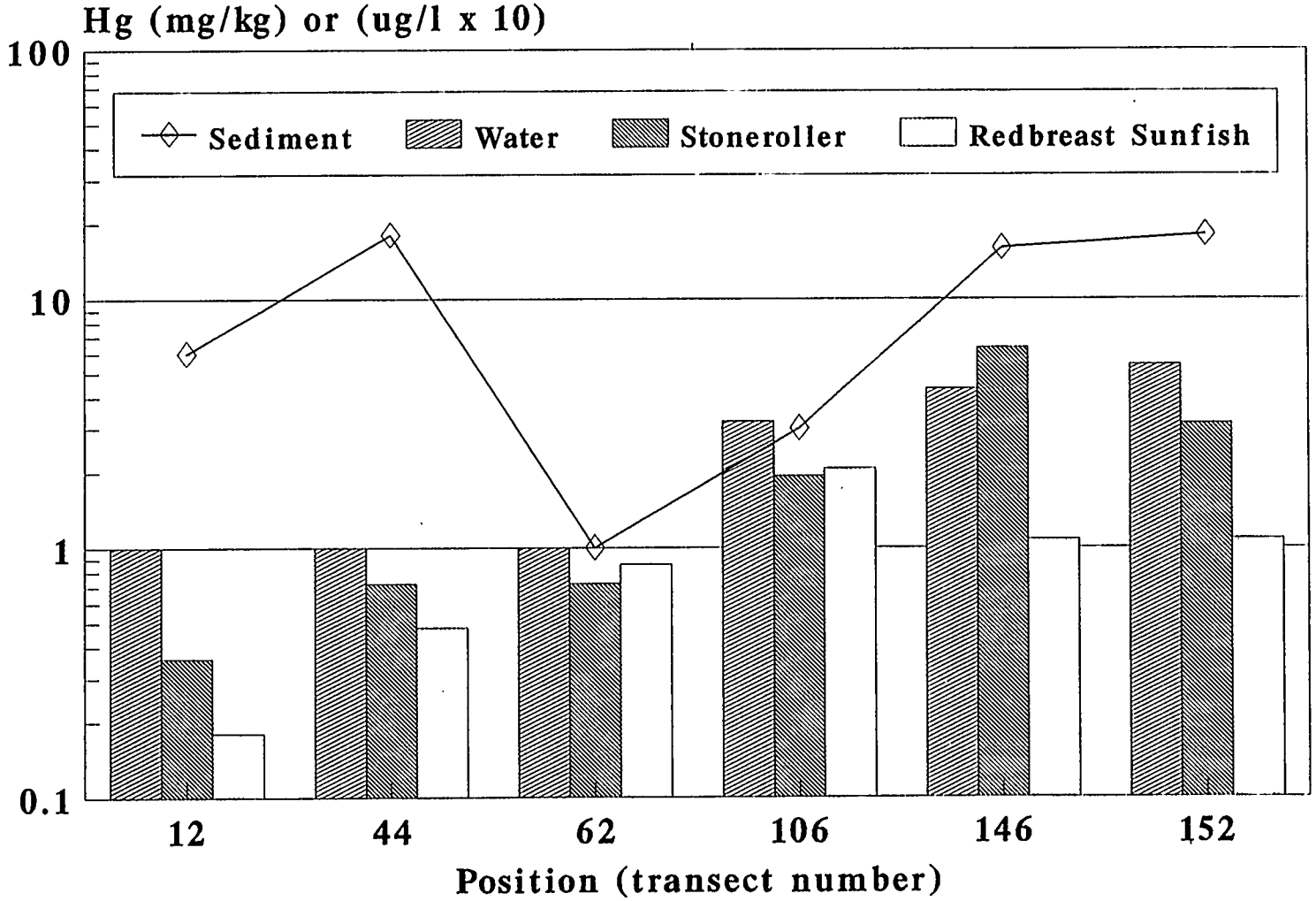

Fig. 6.43. Total mercury concentrations in stonerollers, redbreast sunfish, sediments, and creek water collected from six sampling sites in EFPC, February 10-March 8, 1992.

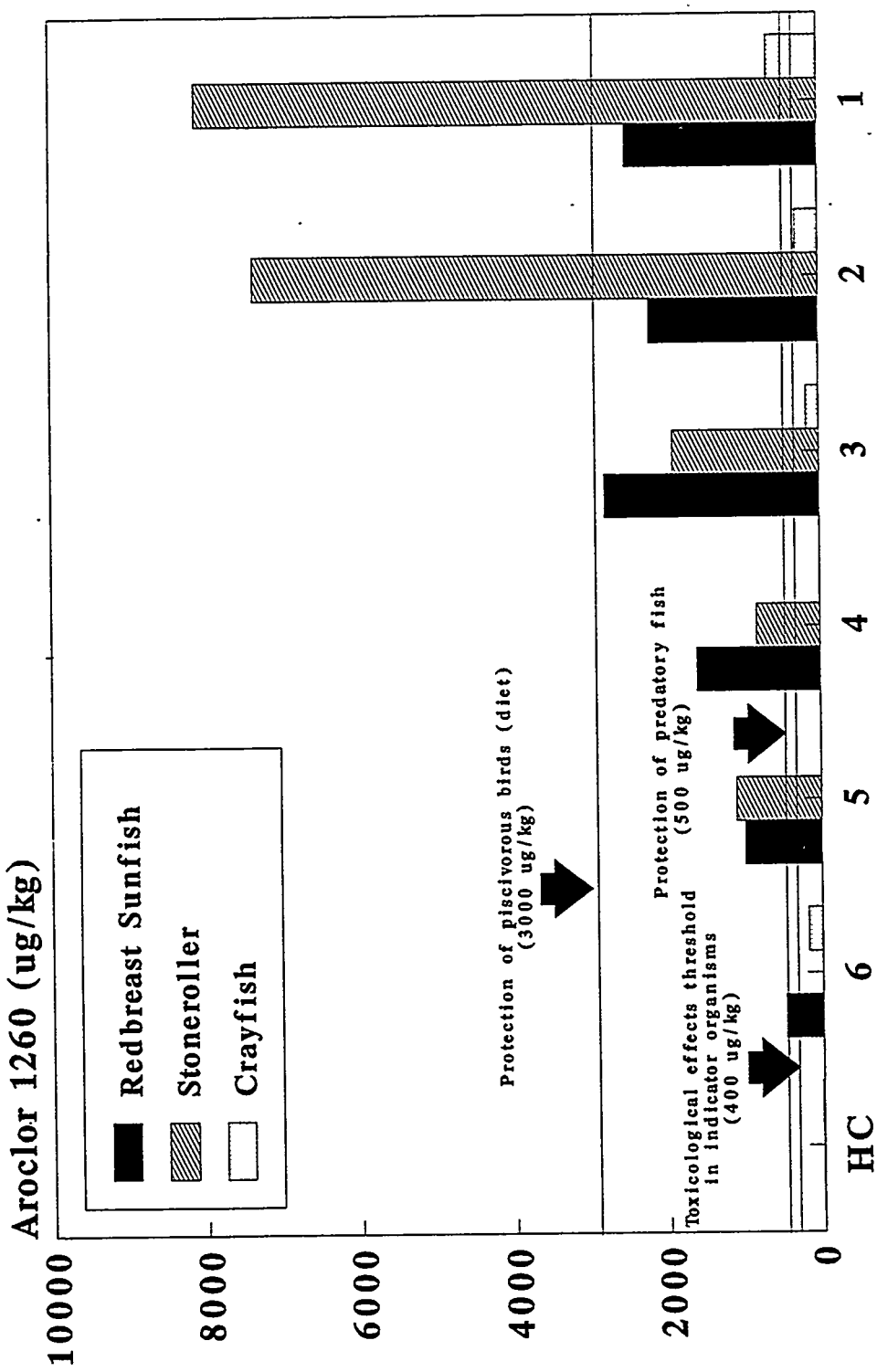

Fig. 6.44. Aroclor 1260 in stonerollers, redbreast sunfish, and crayfish, collected from EFPC and Hinds Creek, October 7-12, 1991. Mean concentrations in stonerollers, redbreast sunfish, and crayfish from Hinds Creek were 3, 7, and 10 μg/kg, respectively.

Aroclor 1260 was undetected in water and sediment samples collected from the six sample sites in EFPC. However, Aroclor 1260 was detected in two composited sediment samples collected between Sites 2 and 3 [from transects 132, 134, and 136 (at 27 mg/kg) and from transects 138, 140, and 142 (at 17 mg/kg)]. These two samples were composited from the first two successive 600-m (1980-ft) reaches downstream from the composited sediment sample that included Site 2. Although Aroclor 1260 concentrations were below detection limits in water and sediment samples, the presence of this PCB mixture in redbreast sunfish and stonerollers indicates that fish collected from these sites had been exposed to Aroclor 1260. The exposure pathways might have been direct contact with water with concentrations that were below the analytical detection limits and/or by ingestion of contaminated food. Interestingly, the concentrations of Aroclor 1260 in redbreast sunfish from the sites in EFPC were strongly positively correlated with the concentrations found in terrestrial insects from the same sites [linear regression, $R^2=0.907$ (Fig. 6.45)].

Pesticides. Mean whole-body concentrations of chlordane, dieldrin, and heptachlor in redbreast sunfish collected from the six sample sites in EFPC followed similar trends. Whole-body concentrations of all three pesticides decreased from those redbreast sunfish collected at Site 1 to those collected at Site 2, increased in fish collected through Site 4 (for chlordane) or Site 5 (for dieldrin and heptachlor), and decreased in fish collected from Site 6. Whole-body concentrations in redbreast sunfish from EFPC ranged from 0.023 to 0.068 mg/kg for chlordane, from 0.020 to 0.067 mg/kg for dieldrin, and from 0.006 to 0.069 mg/kg for heptachlor. Maximum pesticide concentrations were usually obtained in redbreast sunfish collected from Site 5 (Site 4 for chlordane). The whole-body concentrations of chlordane, dieldrin, and heptachlor in redbreast sunfish from the reference site (0.002, 0.013, and 0.006 mg/kg, respectively) were generally less than any of the redbreast sunfish concentrations from EFPC (except for heptachlor at Site 2).

Whole-body concentrations of chlordane, dieldrin, and heptachlor in stonerollers collected from five sample sites in EFPC followed similar trends, but the overall trend was dissimilar to that observed in redbreast sunfish (Fig. 6.46). In stonerollers, the maximum whole-body pesticide concentrations were observed closest to the Y-12 Plant, except for chlordane, which was greatest at Site 2. The general trend for pesticide concentrations in stonerollers was a downstream decrease from the Y-12 Plant to Site 4 (Site 3 for heptachlor), then an increase at Site 5 (heptachlor concentrations decreased downstream to Site 3, then increased at Sites 4 and 5). Mean whole-body concentrations of dieldrin and heptachlor in stonerollers from Sites 4 and 5 were approximately threefold less than the concentrations in redbreast sunfish collected from

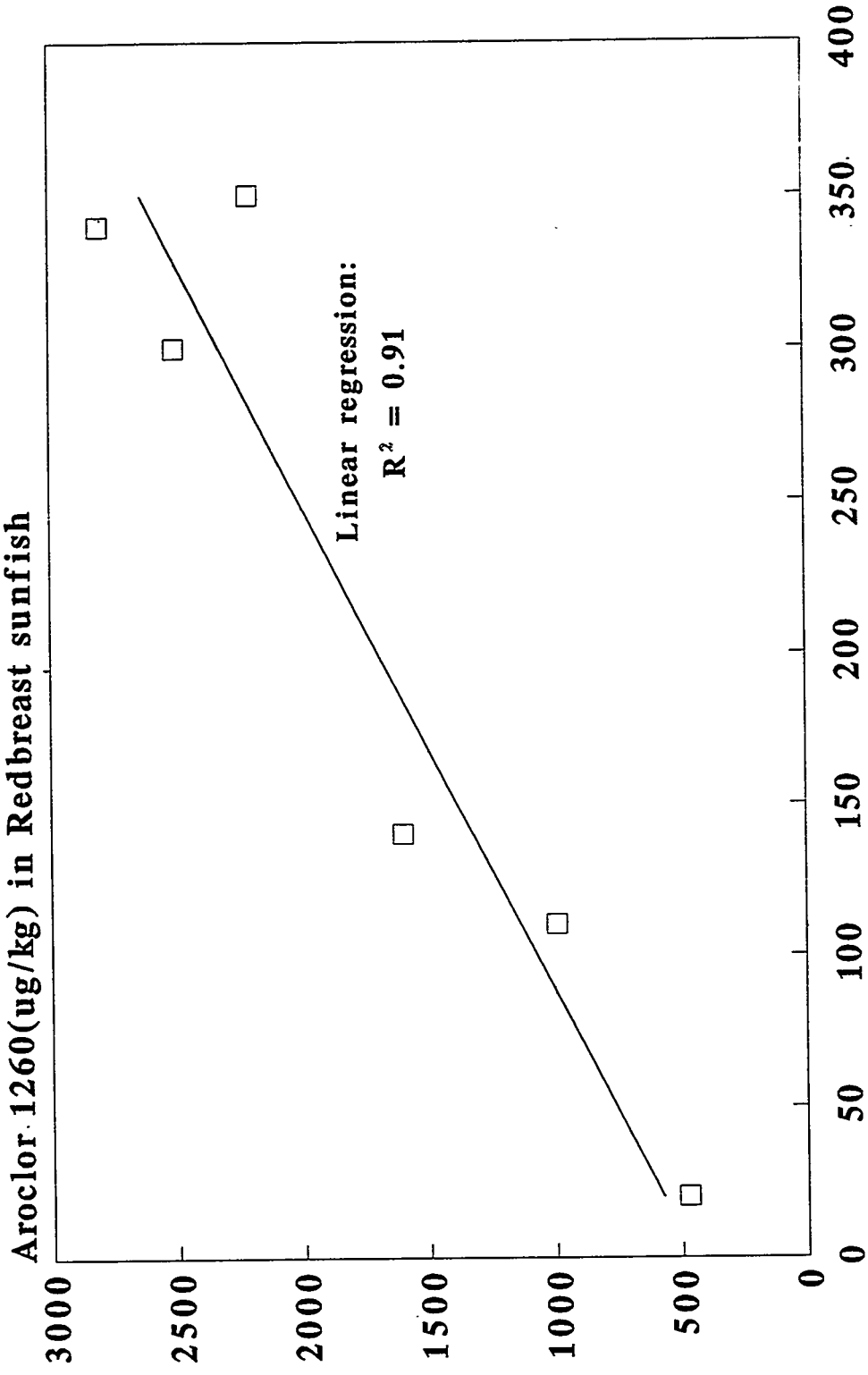

Fig. 6.45. Relationship between total whole-body Aroclor 1260 concentrations in redbreast sunfish and terrestrial insect samples collected from Sites 1 through 6 in EFPC.

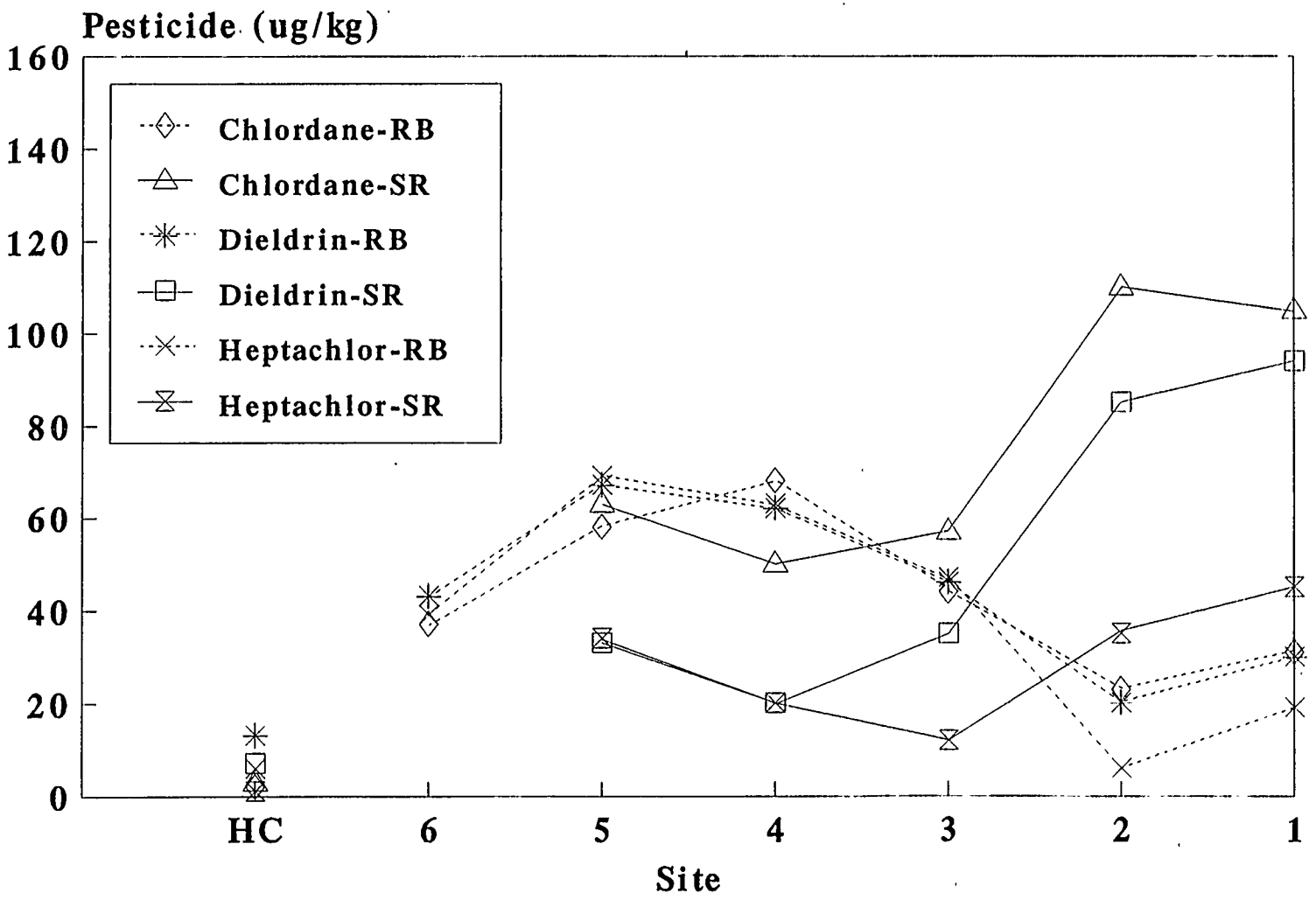

Fig. 6.46. Chlordane, dieldrin, and heptachlor pesticide concentrations in whole-body composite samples of redbreast sunfish and stonerollers collected from EFPC and Hinds Creek, October 7–12, 1991.

the same sites. Whole-body concentrations of chlordane ranged from 0.050 to 0.110 mg/kg, of dieldrin from 0.020 to 0.094 mg/kg, and of heptachlor from 0.0120 to 0.045 mg/kg in stonerollers from EFPC. The maximum concentrations of chlordane and dieldrin in stonerollers collected from EFPC were nearly double the maximum concentration of these two pesticides in redbreast sunfish collected from EFPC. The whole-body concentrations of chlordane, dieldrin, and heptachlor in stonerollers from the reference site (0.003, 0.006, and 0.001 mg/kg, respectively) were less than any of the stoneroller samples collected from EFPC.

PAHs. In redbreast sunfish, PAHs were usually below detection limits (ranging from 0.001 to 0.051 mg/kg). One exception, acenaphthene, ranged from whole-body concentrations of 0.014 mg/kg in redbreast sunfish at Site 6 to 0.018 mg/kg at Site 1, in contrast with the 0.008 mg acenaphthene/kg found in redbreast sunfish from the reference site.

Whole-body PAH concentrations in stonerollers from EFPC were usually greatest at Site 1, closest to the Y-12 Plant, and decreased downstream (Fig. 6.47). Two notable exceptions were naphthalene and phenanthrene. Naphthalene concentrations in stonerollers were 0.021 mg/kg at Site 1, increased to 0.115 mg/kg at Site 3, decreased to 0.054 mg/kg at Site 4, then increased to 0.38 mg/kg at Site 5. Only Site 5 exceeded the 0.241 mg/kg, whole-body naphthalene average concentration in stonerollers at the reference site. Phenanthrene whole-body concentrations in stonerollers ranged from 0.028 mg/kg at Site 1 to 0.042 mg/kg at Site 3, and were greater than the concentration observed for stonerollers collected from Hinds Creek (0.001 mg/kg).

Other inorganics. Whole-body uranium concentrations in stonerollers were greatest near the Y-12 Plant (at 0.85 mg/kg), and decreased from 0.85 mg/kg at Site 1 to 0.052 mg/kg at Site 6 (Fig. 6.48). Uranium concentrations in stonerollers collected from Sites 1 through 4 all exceeded the concentration of uranium in stonerollers collected at the reference site. The pattern of decreasing uranium concentrations in stonerollers downstream from the Y-12 Plant suggests that the plant is the probable uranium source and that stonerollers are exposed through ingestion of periphyton contaminated with uranium through deposition, direct uptake, or both.

Uranium concentrations in redbreast sunfish from all sites in EFPC and the reference site were below the detection level, which ranged from 0.253 mg/kg at Site 2 to 0.063 mg/kg at Site 6. Uranium is not typically bioconcentrated in organisms at higher trophic levels due primarily to low assimilation efficiencies (Swanson 1985). In addition, uranium does not display a great propensity to bioaccumulate in fish tissues (Poston 1982). Therefore, the observed results in redbreast sunfish do not appear to be unusual.

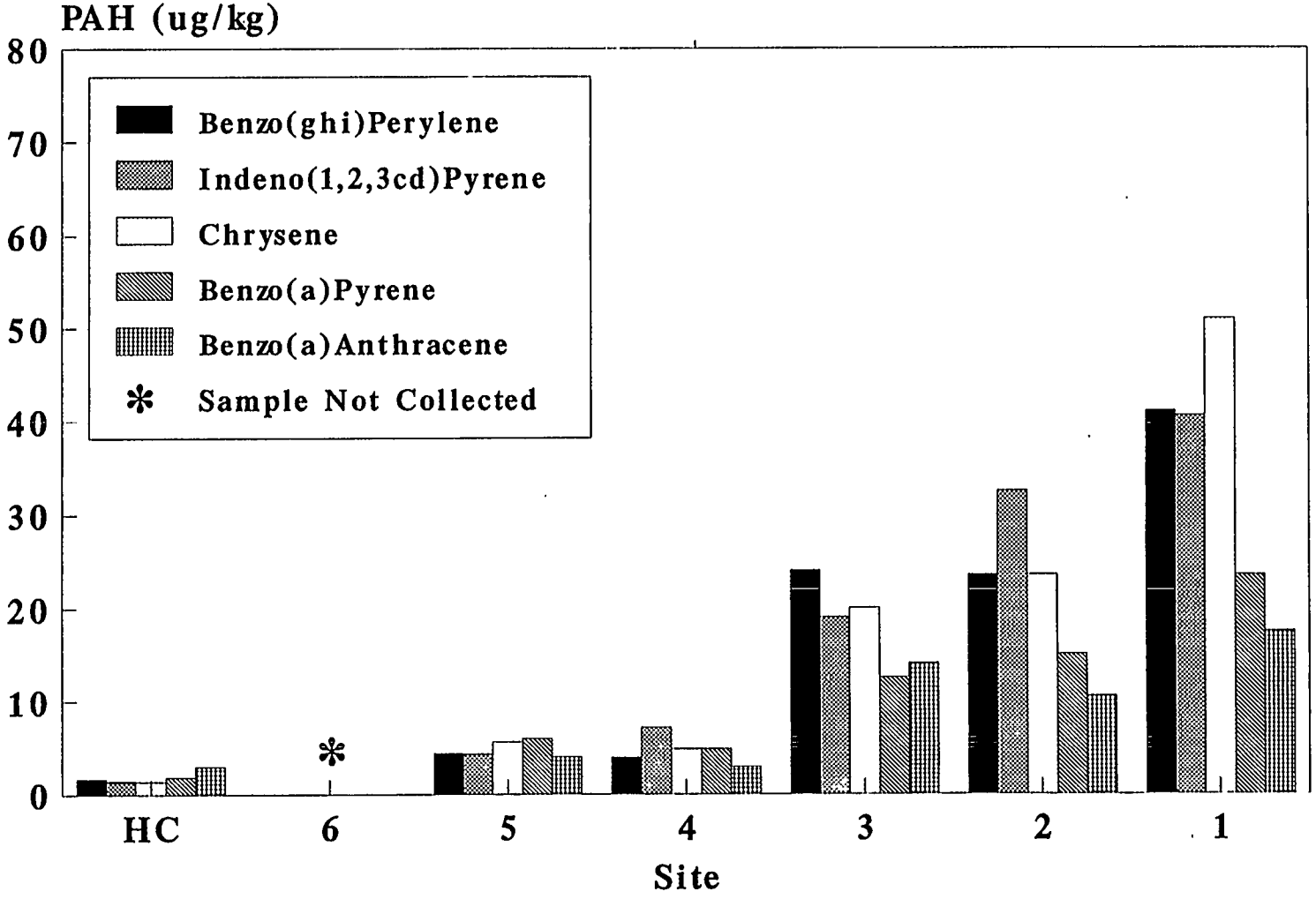

Fig. 6.47. Whole-body concentrations of various PAHs in stonerollers collected from EFPC and Hinds Creek, October 7–12, 1991.

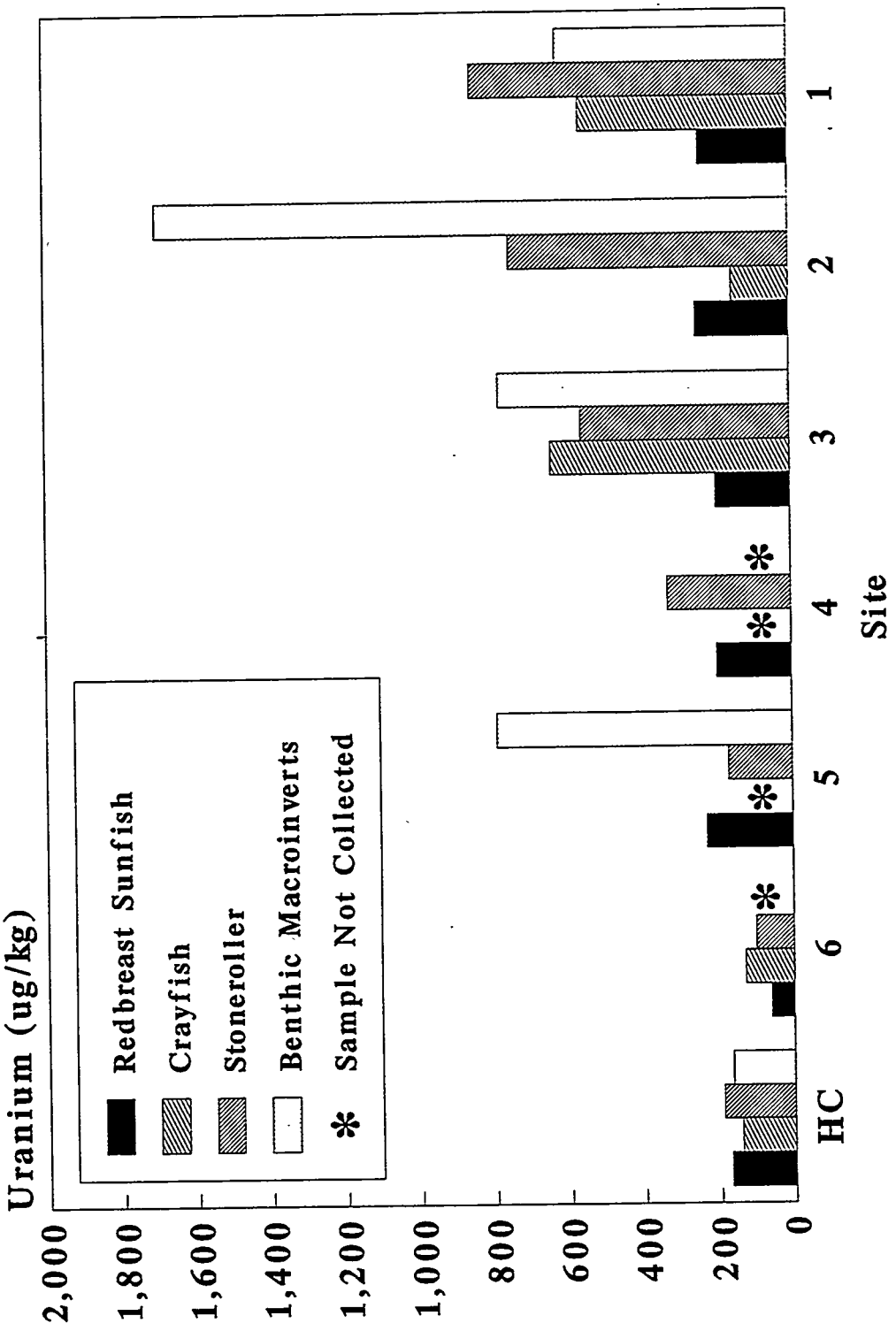

Fig. 6.48. Uranium concentrations in whole-body composite samples of aquatic biota collected from EFPC and Hinds Creek, October 7-29, 1991.

Benthic macroinvertebrates

Sampling methods and population survey results for benthic macroinvertebrates are presented and discussed in Sect. 6.3.3.2.

Mercury. Sufficient sample mass for mercury analysis was collected at four sites in EFPC (Sites 1, 2, 3, and 5) and at the reference site (Fig. 6.49). The maximum whole-body mercury concentration among the composited samples of benthic macroinvertebrates from EFPC was 13.74 mg/kg, obtained from Site 2. Mercury concentrations ranged from 1.40 to 1.71 mg/kg among the benthic macroinvertebrate samples collected from the three other sites in EFPC, and did not increase or decrease in a consistent pattern. However, the patterns of whole-body mercury concentrations in benthic macroinvertebrates and stonerollers collected from the first three sites downstream from the Y-12 Plant were very similar (Figs. 6.41 and 6.49). Mercury concentration in the benthic macroinvertebrate sample from Hinds Creek was below the detection level of 0.25 mg/kg—less than the levels in all four samples collected from EFPC.

Whole-body concentrations of mercury in benthic macroinvertebrates collected from the three sites closest to the Y-12 Plant were poorly correlated with total mercury in water or sediments at these sites (linear regression, R^2=0.022 and 0.410, respectively). Possible explanations for the poor correlations are (1) the total mercury concentrations may not equal concentrations available for uptake, (2) water-borne mercury may represent a minor exposure pathway compared to ingestion for the benthic macroinvertebrates, (3) the single sample for water and sediment may have provided an inaccurate estimate of mercury exposures that actually occur over longer periods due to fluctuations in contaminant concentrations, or (4) water and sediment mercury concentrations may not equal mercury concentrations in the creek-bed boundary layer or pore water where the sampled benthic macroinvertebrates reside.

PCBs. Aroclor 1260 was the only PCB mixture present above the detection limit in the EFPC samples. The greatest whole-body concentration of Aroclor 1260 in benthic macroinvertebrates— 0.60 mg/kg—was observed in the composite sample collected closest to the Y-12 Plant at Site 1 (Fig. 6.50). Aroclor 1260 concentration in samples collected from Site 2 was 0.11 mg/kg, nearly a sixfold decrease in concentration compared with Site 1. Insufficient sample mass was collected from the reference site for comparison.

Pesticides. Sufficient sample mass for the analysis of the organic contaminants was obtained only at the two sites closest to the Y-12 Plant (Sites 1 and 2). Most pesticide concentrations in the benthic macroinvertebrate samples were below the detection limit

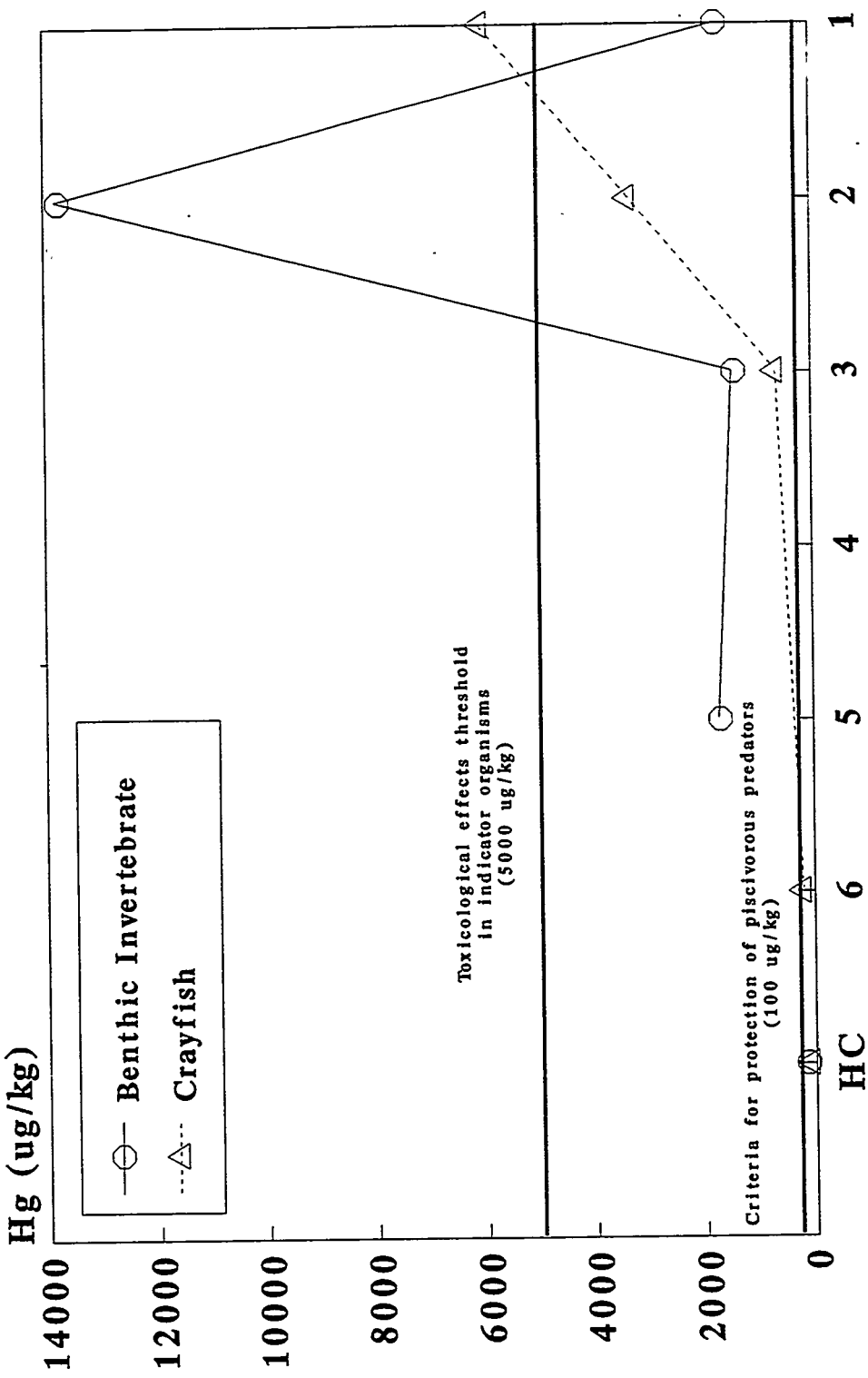

Fig. 6.49. Mercury concentrations in whole-body composite samples of benthic macroinvertebrates and crayfish collected from EFPC and Hinds Creek, October 7-29, 1991.

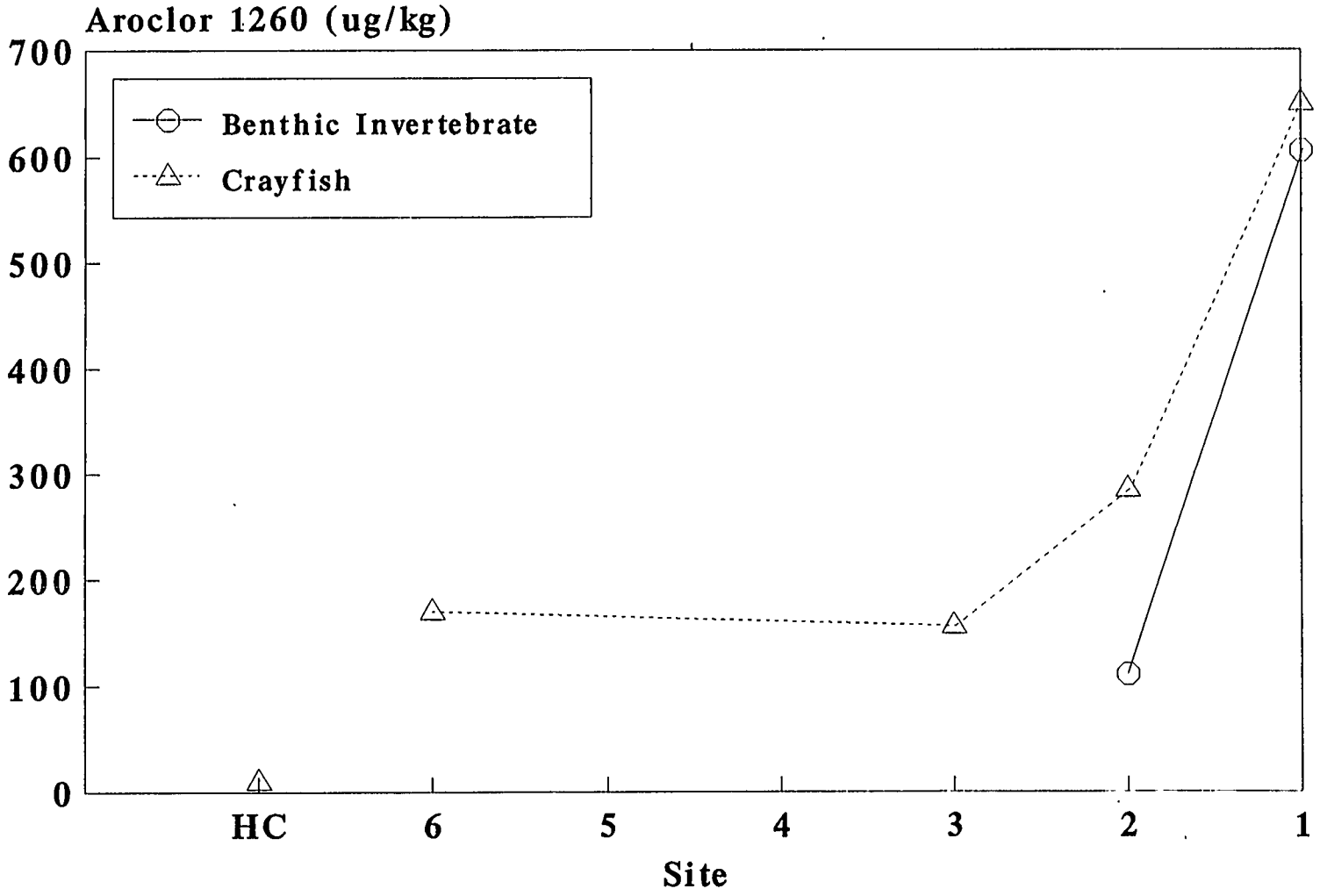

Fig. 6.50. Aroclor 1260 whole-body concentrations in benthic macroinvertebrates and crayfish collected from EFPC and Hinds Creek, October 7–29, 1991.

(Table 6.24). The pesticides detected in the benthic macroinvertebrate samples were typically present in low concentrations (e.g., 0.002 mg/kg heptachlor at Site 1).

PAHs. The concentrations of most individual PAHs were similar in benthic macroinvertebrate samples collected from either Site 1 or Site 2 (Table 6.24), and no distinct trend was evident in sample PAH concentrations downstream from the Y-12 Plant.

Other inorganics. Whole-body uranium concentrations among the benthic macroinvertebrates were highest in the sample collected from Site 2 (at 1.70 mg/kg) and were 0.62, 0.78, and 0.80 mg/kg in samples collected from Sites 1, 3, and 5, respectively (Fig. 6.48). Uranium concentration in the benthic macroinvertebrate sample from Hinds Creek was below the detection level (0.33 mg/kg). This pattern is similar to that observed for mercury.

Crayfish

Mercury. Whole-body mercury concentrations in crayfish collected from the four sample sites in EFPC with sufficient sample mass ranged from 6.02 to 0.23 mg mercury/kg, but the sample collected from Hinds Creek was below the detection limit (0.20 mg/kg) (Fig. 6.49). The largest whole-body mercury concentrations in crayfish were obtained at the site closest to the Y-12 Plant and decreased downstream. Whole body mercury concentrations in crayfish from all sample sites in EFPC exceeded those from the reference site.

Whole-body mercury concentrations in crayfish collected from the three sites closest to the Y-12 Plant exhibited a high positive correlation with total mercury concentrations in water and in sediments (linear regression, $R^2 = 0.999$ and 0.920, respectively).

PCBs. Aroclor 1260 was the only PCB mixture present above the sample detection limit in crayfish, just as it was the only PCB mixture detected in redbreast sunfish, stonerollers, and benthic macroinvertebrates. Sufficient sample mass of crayfish was collected for PCB analyses at four sites in EFPC (Sites 1, 2, 3, and 6) as well as at the reference site (Fig. 6.50). The greatest whole-body concentration of Aroclor 1260 in crayfish from EFPC (0.65 mg/kg) was observed in the composite sample collected nearest the Y-12 Plant at Site 1. The concentrations then decreased downstream to Site 3, but increased slightly at Site 6 (Fig. 6.50). Aroclor 1260 concentrations in crayfish collected from every site in EFPC exceeded the concentrations observed in crayfish collected from Hinds Creek (0.01 mg/kg).

Pesticides. Sufficient sample mass was collected for pesticide analyses at Sites 1, 2, and 3 in EFPC, and at the reference site (Fig. 6.51). Whole-body concentrations of chlordane,

dieldrin, and DDE were each greatest in crayfish closest to the Y-12 Plant (at 0.090, 0.010, and 0.011 mg/kg, respectively) and probably decreased downstream (Fig. 6.51). Pesticide concentrations in crayfish from the reference site were either below the detection limit (e.g., < 0.039 mg/kg for dieldrin) or less than the levels observed in samples collected from EFPC (e.g., < 0.009 mg/kg chlordane in Hinds Creek crayfish versus 0.021 to 0.09 mg/kg in EFPC crayfish samples).

PAHs. Sufficient crayfish mass was collected for PAH analyses at three sites in EFPC (Sites 1, 2, and 3) and at Hinds Creek (Fig. 6.52). Whole-body concentrations of each of the 16 individual PAHs were all at less than 0.05 mg/kg in crayfish samples collected from all sample sites. The whole-body concentration of nearly every individual PAH increased downstream from the Y-12 Plant, reaching maximum concentrations in crayfish collected from Site 3 (Fig. 6.52). In addition, the whole-body concentrations of most individual PAHs in crayfish collected from the reference site were ~2- to 22-fold less than concentrations in crayfish collected from the three sites in EFPC.

The pattern of increased body burden of PAHs in crayfish downstream from the Y-12 Plant is opposite to the pattern of decreased PAHs in stonerollers collected from the same locations.

Other inorganics. Uranium concentrations in crayfish composite samples collected from Sites 1 and 3 were 0.77 and 0.64 mg/kg, respectively. In contrast, uranium concentrations in crayfish collected from other sites in EFPC and Hinds Creek were below the sample detection limits.

Flying insects with aquatic juveniles

Flying insects whose juvenile life stages are predominantly aquatic were composited for analysis. Means and standard deviations of the results were calculated by assuming that samples reported as below detection limits contained the contaminant at the sample detection limit.

Mercury. Concentrations of mercury in aquatic insects are presented in Table 6.26. The reference sample was reported as below the sample detection limit of 168 μg/kg, and the sample from Site 6 was below the sample detection limit of 126 μg/kg. Other sites ranged from 370 μg/kg to 703 μg/kg. Mercury concentrations in aquatic insect composites were not correlated statistically with sediment mercury concentrations.

Other inorganics. Antimony was above sample detection limits in four samples and above the reference level at Site 2 (Table 6.26). Arsenic was detected in three samples, with a

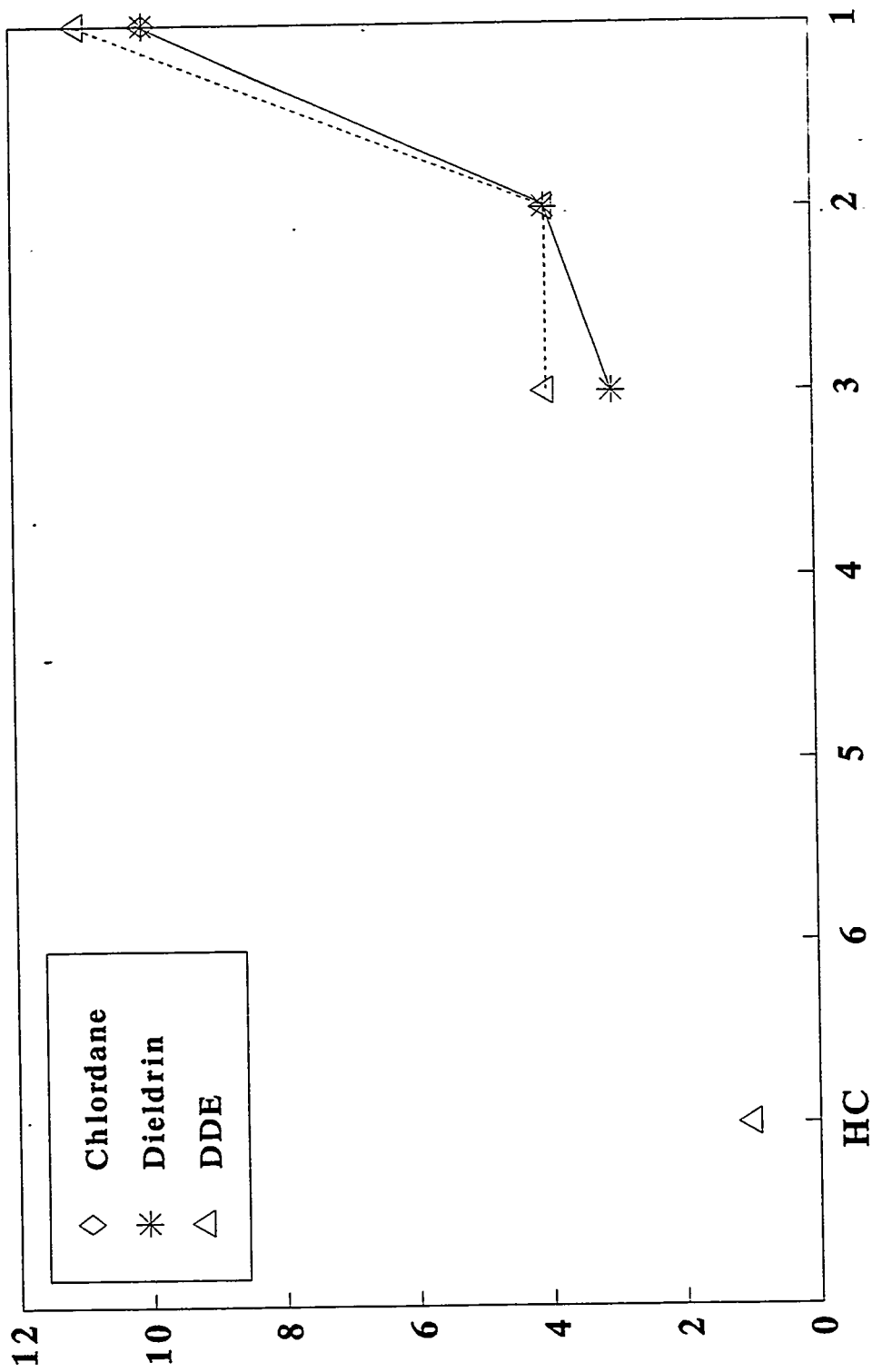

Fig. 6.51. Chlordane, dieldrin, and DDE pesticide whole-body concentration in crayfish collected from EFPC and Hinds Creek, October 7-12, 1991.

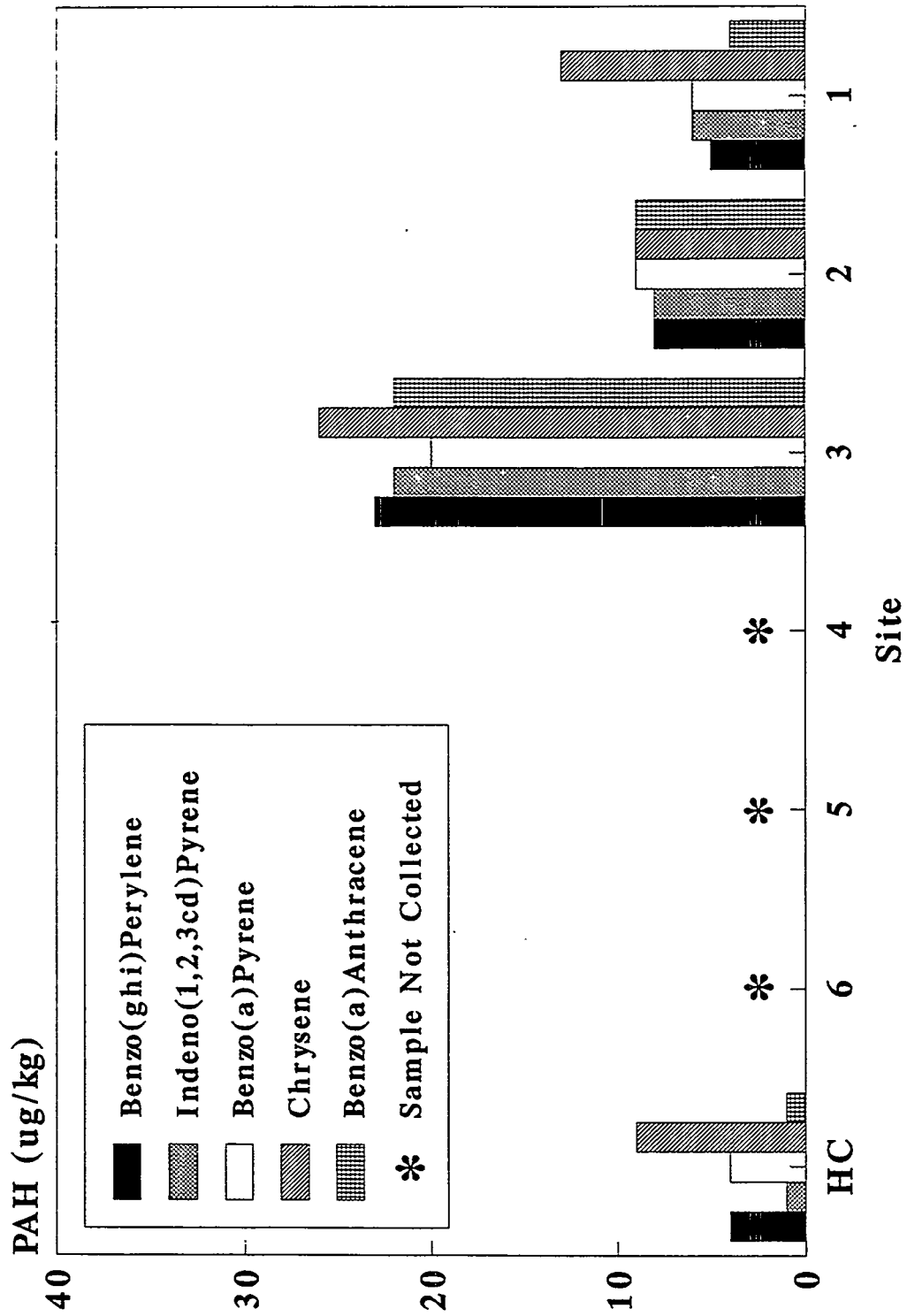

Fig. 6.52. Concentrations of various PAHs in whole-body composite crayfish samples from EFPC and Hinds Creek, October 7-12, 1991.

Table 6.26. Concentrations of inorganic analytes in aquatic insects at EFPC and Hinds Creek[a]

Analyte	1	2	3	4	5	6	Reference
Mercury	538	351	443	370	703	<126.1	<168.4
Antimony	21	97	<7.2	10	<11.4	27	65
Arsenic	<155.7	<108.8	120	621	<227.4	191	190
Cadmium	<1,008.4	<1,058.8	<970.5	<863.3	<1,883.1	743	<954.3
Chromium	395	786	531	1,385	799	623	265
Zinc	82,855	69,303	72,033	96,905	85,173	112,830	87,422
Selenium	768	1,082	909	1,520	824	594	577
Uranium	<159	<173.6	<119.9	<110.7	<272.1	<74.4	<149.7

[a] < indicates the analyte concentration was below the detection limit.

maximum at Site 4 of about three times the reference level. Cadmium was below sample detection limits in all samples except Site 5, which was below the sample detection limit of the reference sample. All chromium concentrations were above the concentration in the reference sample, peaking at Site 4 at five times the reference concentration. Selenium was above detection limits and above the concentration in the reference sample at all sample sites. Uranium was below sample detection limits in all samples. Zinc was above sample detection limits in all samples and above the concentration in the reference sample at Sites 4 and 6.

PCBs, pesticides, and PAHs. Insufficient quantities of samples were recovered to allow analysis of organic contaminants.

Mice

Sampling methods and population survey results for small mammals are discussed in Sect. 6.3.3.5. Mice were collected from live traps after the population survey was completed. Individual whole mice were homogenized for analysis.

Mercury. Analytical results for mercury are summarized in Table Q.2 (Appendix Q), and average values are listed in Table 6.27. Mercury was reported as below the sample detection limit (~200 to 400 μg/kg) in nine of eleven individuals. Two individuals at Site 2 had mercury levels above the sample detection limits, at 675 and 1105 μg mercury/kg, respectively.

PCBs. Analytical results for PCBs are given in Table Q.2 (Appendix Q), and average values are given in Table 6.28. Aroclor 1260 was detected above sample detection limits in all samples except those from the reference site. All other PCB mixtures were reported as below sample detection limits in all homogenates. Aroclor 1260 concentrations ranged from 57 μg/kg to 480 μg/kg. The highest concentration was at Site 1 and the second highest was at Site 4, with lower values intervening.

Pesticides. Few pesticides were detected above method detection limits (Table 6.29). One value for DDT and one value for heptachlor epoxide were reported as more than 4 times the detection limit; the remainder of the reported values were estimated below the detection limit (J-qualified).

PAHs. Ten mouse homogenates were analyzed for 16 PAH compounds each. Of the 160 results, 44 were estimated below the detection limit (J-qualified). The remainder were below the sample detection limit (Table Q.2, Appendix Q.)

Table 6.27. Inorganics in mouse homogenate from EFPC and Mill Branch[a]

Analyte	Sampling Site						
	1	2	3	4	5	6	Reference
Antimony	160	101	13	164	80	252	<17
Arsenic	<262	<228	<286	<179	<225	<158	<335
Cadmium	<2041	<1747	<2166	<1536	<2034	<1352	<2736
Chromium	371	1142	1944	260	1333	<331	<468
Mercury	<331	888	<335	<229	<311	<196	<376
Selenium	563	371	742	314	<476	<493	<608
Uranium	<294	<265	N.A.[b]	<218	<304	<212	<395
Zinc	34811	43649	32762	32713	44482	23739	35386

[a]Averages of analytical results in μg/kg.
[b]N.A. = Not analyzed

Table 6.28. PCBs in mouse homogenate from EFPC and Mill Branch[a]

Sample site	1	2	3	4	5	6	Reference
Aroclor 1260	445	114	160	335	79	71	<20
Aroclor 1254	<12	<12	<12	<12	<12	<12	<12
Aroclor 1248	<60	<60	<60	<60	<60	<60	<60
Aroclor 1242	<60	<60	<60	<60	<60	<60	<60
Aroclor 1232	<60	<60	<60	<60	<60	<60	<60
Aroclor 1221	<120	<120	<120	<120	<120	<120	<120
Aroclor 1016	<60	<60	<60	<60	<60	<60	<60

[a]Concentrations in μg/kg

Table 6.29. Pesticides in mouse homogenate from EFPC and Mill Branch[a]

Sample site	1	2	3	4	5	6	Reference
Aldrin	1	<3	1	1	<6	<6	<6
Alpha-Chlordane	0	1	1	1	2	1	1
Alpha-BHC	4	<1	<16	1	1	1	1
Beta-BHC	<54	<54	<54	17	14	<54	<54
DDD	<22	<22	<22	<22	<22	<22	<22
DDE	1	1	1	1	8	1	1
DDT	9	13	9	<14	<5	<14	9
Delta-BHC	3	<8	<8	<8	<8	<8	<8
Dieldrin	<39	10	<39	<39	10	<39	<39
Alpha-Endosulfan	<14	<14	<14	<14	<14	<14	<14
Endosulfan	<17	<17	<17	<17	<17	<17	0
Endrin	<25	<25	<25	<25	<25	<25	<25
Endrin aldehyde	<51	<51	<51	<51	<51	<51	<51
Endrin ketone	<16	<16	<16	<16	<16	<16	<16
Endosulfan sulfate	<19	<19	<19	<19	<19	<19	<19
Gamma-BHc	5	<15	<15	<6	<15	<15	2
Gamma-Chlordane	4	4	<14	4	<14	<14	<14

Table 6.29 (continued)

Sample site	1	2	3	4	5	6	Reference
Heptachlor	<16	<16	<16	<16	<16	<16	<16
Heptachlor epoxide	3	5	5	7	3	3	3
Methoxychlor	<150	<150	<150	<150	<150	<150	<150
Toxaphene	<150	<150	<150	<150	<150	<150	<150

[a]Concentrations in μg/kg

Other inorganics. Analytical results for other inorganic analytes are given in Table Q.2 (Appendix Q) and Table 6.27. Arsenic and uranium were below sample detection limits in all mouse homogenates. Cadmium and zinc were reported at less than the reference concentration in all samples. Antimony was not detected in the reference sample, but it was reported at concentrations above the reference sample detection limit in six of ten EFPC floodplain samples. The individual maxima of 313 μg/kg occurred at Sites 1 and 4, but the second homogenate from each of these sites was near the detection limit. As a result, the maximum site average, 194 μg/kg, occurred at Site 5, where both individuals had moderate but similar values.

Chromium was not detected in the reference sample, but it was reported above sample detection limits in eight of ten EFPC floodplain samples. The maximum chromium value reported was 2108 μg/kg at Site 5. One of two other individuals at that site had a detectable concentration of 558 μg/kg; the second was reported as below sample detection limits. Selenium was not detected in the reference sample, but it was detected in five of ten EFPC floodplain samples, at concentrations ranging from 353 μg/kg to 772 μg/kg. The highest concentrations were observed at Sites 1 and 3.

Shrews

Shrews were captured at Sites 4 and 5 only. Analytical results are presented in Table Q.2 (Appendix Q) and in Table 6.30.

Mercury. Mercury was reported at 1.9 mg/kg and 7.9 mg/kg at Sites 4 and 5, respectively.

PCBs. Aroclor 1260 was detected at 950 μg/kg and 1400 μg/kg at Sites 4 and 5, respectively. Other PCB mixtures were not detected.

Pesticides. One pesticide analyte was reported at more than four times the sample detection limit and qualified because of interferences; five were estimated below detection limits in at least one sample, and the remainder were not detected in either sample.

PAHs. PAHs were not analyzed in these homogenates. Mean and maximum concentrations of antimony and chromium were less than in mouse homogenates, and concentrations of selenium and zinc were similar to those in mice, whereas arsenic, cadmium, and mercury levels were above those in mice.

Other inorganics. Uranium was not detected at sample detection limits.

Table 6-30. Results of analysis of infrequently captured biota from the EFPC floodplain[a]

ANALYTE	Shrew Site 4	Shrew Site 5	Vole Site 4	Heron feather Site 6	Heron liver Site 6	Wren Site 2	Wren Site 4
Aroclor-1260	1400	950	46	b	1400	1300	2900
Aroclor-1254	<36	<24	<12	b	<60	<120	<140
Aroclor-1248	<180	<120	<60	b	<300	<600	<690
Aroclor-1242	<180	<120	<60	b	<300	<600	<690
Aroclor-1232	<180	<120	<60	b	<300	<600	<690
Aroclor-1221	<360	<240	<120	b	<600	<1200	<1400
Aroclor-1016	<180	<120	73	b	<300	<600	<690
Acenaphthylene	b	b	4	b	310	<630	b
Acenaphthene	b	b	37	b	<240	21	b
Anthracene	b	b	19	b	10	16	b
Benzo(a)pyrene	b	b	50	b	1	2	b
Benzo(b)fluoranthene	b	b	55	b	<3	1	b
Benzo(k)fluoranthene	b	b	30	b	1	2	b
Benzo(a)thracene	b	b	27	b	<2	1	b
Benzo(g,h,i)perylene	b	b	41	b	<7	<14	b
Chrysene	b	b	73	b	<18	<36	b
Dibenzo(a,h)anthracene	b	b	7	b	<3	<5	b
Fluoranthene	b	b	53	b	<30	<59	b
Fluorene	b	b	<41	b	<33	<65	b
Indeno(1,2,3-cd)pyrene	b	b	62	b	0	<16	b
Napthalene	b	b	63	b	10	4	b
Phenanthrene	b	b	6	b	<93	<180	b
Pyrene	b	b	<45	b	<36	<71	b
Aldrin	3	<13	<6	b	8	<63	10
Alpha-chlordane	<50	8	0	b	12	30	1

Table 6.30 (continued)

ANALYTE	Shrew Site 4	Shrew Site 5	Vole Site 4	Heron feather Site 6	Heron liver Site 6	Wren Site 2	Wren Site 4
Alpha-BHC	<48	<32	1	b	4	<160	<180
Beta-BHC	<160	<110	23	b	<270	<540	<620
DDD	<67	<44	<22	b	<110	<220	<250
DDE	17	14	2	b	180	770	25
DDT	<14	<14	7	b	<70	95	<16
Delta-BHC	4	3	2	b	<38	<75	14
Dieldrin	8	12	1	b	130	<390	38
Alpha-Endosulfan	<40	<27	<14	b	<68	<140	<150
Beta-Endosulfan Endo-SII	<52	<35	<17	b	<87	<170	<200
Endrin	<74	<49	<25	b	<120	<250	<280
Endrin-aldehyde	<150	<100	<51	b	<260	<510	<580
Endrin-ketone	<48	<32	<16	b	<80	<160	<180
Endrin sulfate	<56	<37	<19	b	<93	<190	<210
Gamma-BHC	4	<30	2	b	<75	<150	<170
Gamma-Chlordane	<42	<28	<14	b	<70	<140	<160
Heptachlor	<48	3	<16	b	<80	<160	<180
Heptachlor-Epoxide	19	28	<9	b	41	260	28
Methoxychlor	<460	<310	<150	b	<760	<1500	<1700
Toxaphene	<450	<300	<150	b	<750	<1500	<1700
Mercury	1911	7890	<294	5302	8986	3486	3550
Antimony	116	35	64	<26	<88	321	77
Arsenic	344	766	<273	<484	<143	<113	<269
Cadmium	5949	<2024	<2021	<3236	<1445	<1042	4669
Chromium	567	526	1265	7993	<209	604	<357

Table 6.30 (continued)

ANALYTE	Shrew Site 4	Shrew Site 5	Vole Site 4	Heron feather Site 6	Heron liver Site 6	Wren Site 2	Wren Site 4
Selenium	934	1240	<417	<1875	3475	696	1039
Zinc	40798	31496	37991	116200	60124	34858	42168
Uranium	<327	<262	<306	<403	<218	<169	<411

[a]Concentrations in μg/kg
[b]Not analyzed

Vole

Only one vole sample was obtained during the study. It was trapped at Site 4. Analytical results are included in Table 6.30.

Mercury. Mercury was reported at less than the sample detection limit of 294 μg/kg.

PCBs. Aroclor 1260 was reported at 46 μg/kg, and Aroclor 1016 was reported at 73 μg/kg. Both were reported at less than five times the detection limit.

Pesticides. Of 21 pesticide analytes, 8 were estimated at less than the detection limit, whereas 13 were not detected.

PAHs. Of 16 PAH analytes, 14 were reported, but all were qualified.

Other inorganics. Five of seven other inorganic analytes were at less than the sample detection limits. Chromium and zinc were reported at levels similar to those in mice.

Heron

A heron was inadvertently killed when it flew into a net during a survey of bat populations. Its liver and breast feathers were analyzed (Table 6.30). No heron sample from an unaffected location was available as a reference sample.

Mercury. Mercury concentrations in breast feathers and liver were reported as 5.3 mg/kg and 9.0 mg/kg, respectively. These concentrations were above those for whole-body composites of mice, shrews, vole, and wrens, but below composites of earthworms.

PCBs. Aroclor 1260 in liver was reported as 1.4 mg/kg, nearly three times the maximum mouse whole-body concentration and approximately the same as the maximum whole-body concentration in redbreast sunfish, but about one-sixth the maximum level reported in stonerollers. All other PCB mixtures were reported as undetected at the sample detection limits.

Pesticides. Aldrin, α-BHC, α-chlordane, dieldrin, DDE, and heptachlor epoxide were reported; of these, all but 5 were estimated at below the target sample detection limit. Levels of DDE, α-chlordane, and heptachlor epoxide were lower than in homogenates of wren tissue.

PAHs. No PAH analytes were reported as above detection limits in heron liver; 6 of 16 were estimated as less than the sample detection limit. All except naphthalene were lower than the corresponding values for wrens.

Other inorganics. In breast feathers, only chromium and zinc were reported above sample detection limits. In liver, only selenium and zinc were reported at levels above sample detection limits. Chromium in feathers was at more than ten times the concentrations found in whole-body composites of mice or wrens. Zinc in feathers was approximately three times the average concentrations in mice and wrens; zinc in liver was 1.5 to 2 times the average levels in mice and wrens; and selenium in liver was 4 to 6 times the average selenium levels in composites of mice and wrens.

Wrens

Two wrens, one each at Sites 2 and 4, were inadvertently captured in the mammal traps and died. They were included in the analysis as unplanned samples. Results are presented in Table 6.30. There were no wren samples from a reference site.

Mercury. Mercury levels were near 3.5 mg/kg in both samples, more than four times the mean levels in mice and similar to the levels observed in shrews (Table 6.30).

PCBs. - Aroclor 1260 was detected at 1300 μg/kg and 2900 μg/kg. The average of 2100 μg/kg was nearly 10 times the average Aroclor 1260 concentrations in terrestrial insects and mice. No other PCB mixture was detected above sample detection limits.

Pesticides. Of 21 pesticide contaminants, only four (α-chlordane, DDE, DDT, and heptachlor epoxide) were detected. DDE and heptachlor epoxide levels were approximately 5-fold and 10-fold higher, respectively, than in shrews.

PAHs. Only one wren was analyzed for PAHs. Of 16 analytes, 9 were not detected and 7 were estimated at less than the detection limit.

Other inorganics. Arsenic and uranium were reported as below detection limits in both samples, and cadmium and chromium were below detection limits in one sample. Antimony averaged more than 20 times the average level in terrestrial insects; selenium was higher in both samples than in any of the terrestrial insect samples; and the average zinc concentration was less than half the average of terrestrial insects.

Flying insects with terrestrial juveniles

Flying insects were collected in black-light traps and were identified, separated according to the habitat—terrestrial—of their juvenile life stages, and composited for analysis.

Mercury. Analytical results for mercury in insects of terrestrial origin are given in Table 6.31. Mercury was reported as undetected at a sample detection limit of 92 μg/kg in the reference sample and as undetected at sample detection limits of 140 μg/kg at Site 1, 127 μg/kg at Site 4, 119 μg/kg at Site 5, and 91 μg/kg at Site 6. The sample from Site 2 contained 196 μg/kg. The sample at Site 3 was considerably higher, with a reported concentration of 3189 μg/kg. Interestingly, redbreast sunfish from Site 3 also had the highest body burdens of mercury among samples from EFPC.

To determine whether soil and insect body burdens were correlated, a concentration of half the sample detection limit was assumed for samples reported as below detection limits, and surface soil mercury values were averaged for the two sampling transects flanking the insect trap locations. Mercury concentrations in the insect composite samples were not statistically correlated with soil mercury concentrations; however, the samples having the two highest mercury concentrations came from the two sample sites with the most highly contaminated soil.

PCBs. PCB concentrations in terrestrial insects are presented in Table 6.32. Aroclor 1260 concentrations ranged from 20 to 350 μg/kg at EFPC sites; the value at the Hinds Creek reference site was also 20 μg/kg. The concentrations reached a maximum at Site 2 and decreased steadily with distance from the Y-12 Plant (Fig. 6.53). Other PCB mixtures were not reliably identified in the composite samples.

Pesticides. Data for pesticides in terrestrial insect composites are presented in Table 6.33. Neither DDD, DDE, DDT, methoxychlor, α-endosulfan, endrin, nor toxaphene were found above detection limits in the six composite samples. Average levels of aldrin, α-BHC, α-BHC, α-chlordane, β-endosulfan, heptachlor, and heptachlor epoxide were below those at the reference site.

PAHs. PAH levels in insects are summarized in Table 6.34. Six samples were large enough for analysis of PAHs. Acenaphthene was not detected, and seven other analytes were below twice the reference concentration. For seven of the remaining eight analytes, the highest value was reported at Site 3.

Other inorganics. In the reference sample, all inorganics except zinc were below the sample detection limit (Table 6.31); zinc levels in EFPC floodplain samples were less than twice the concentration reported in the reference. Maximum concentrations of antimony, and chromium were found at Site 3. The highest arsenic concentration occurred at Site 4, and the highest selenium concentration was at Site 2. Uranium and cadmium were not detected.

Table 6.31. Inorganic analytes in terrestrial insects homogenates from EFPC and Hinds Creek[a]

Sample site	1	2	3	4	5	6	Reference
Mercury	<140	196	3189	<127	<119	<91	<92
Antimony	7	9	12	10	6	<2	<3
Arsenic	<92	<85	253	292	165	36	<57
Cadmium	<671	<598	<712	<533	<502	<334	<415
Chromium	397	323	730	<239	255	<194	<270
Selenium	<430	572	<411	<283	571	347	<328
Uranium	<81	<74	<81	<60	<63	<42	<53
Zinc	82012	98579	75259	81195	81542	54079	58155

[a]Concentrations in μg/kg

Table 6.32. PCBs in terrestrial insects at EFPC and Hinds Creek[a]

Analyte	Sampling location						
	Site 1	Site 2	Site 3	Site 4	Site 5	Site 6	Reference
PCB1260	300	350	340	140	110	<20	<20
PCB1254	<12	<12	<12	<12	<12	<12	<12
PCB1248	<60	<60	<60	<60	<60	<60	<60
PCB1242	<60	<60	<60	<60	<60	<60	<60
PCB1232	<60	<60	<60	<60	<60	<60	<60
PCB1221	<120	<120	<120	<120	<120	<120	<120
PCB1016	590	<60	<60	<60	<60	230	<60

[a]Analytical results in μg/kg.

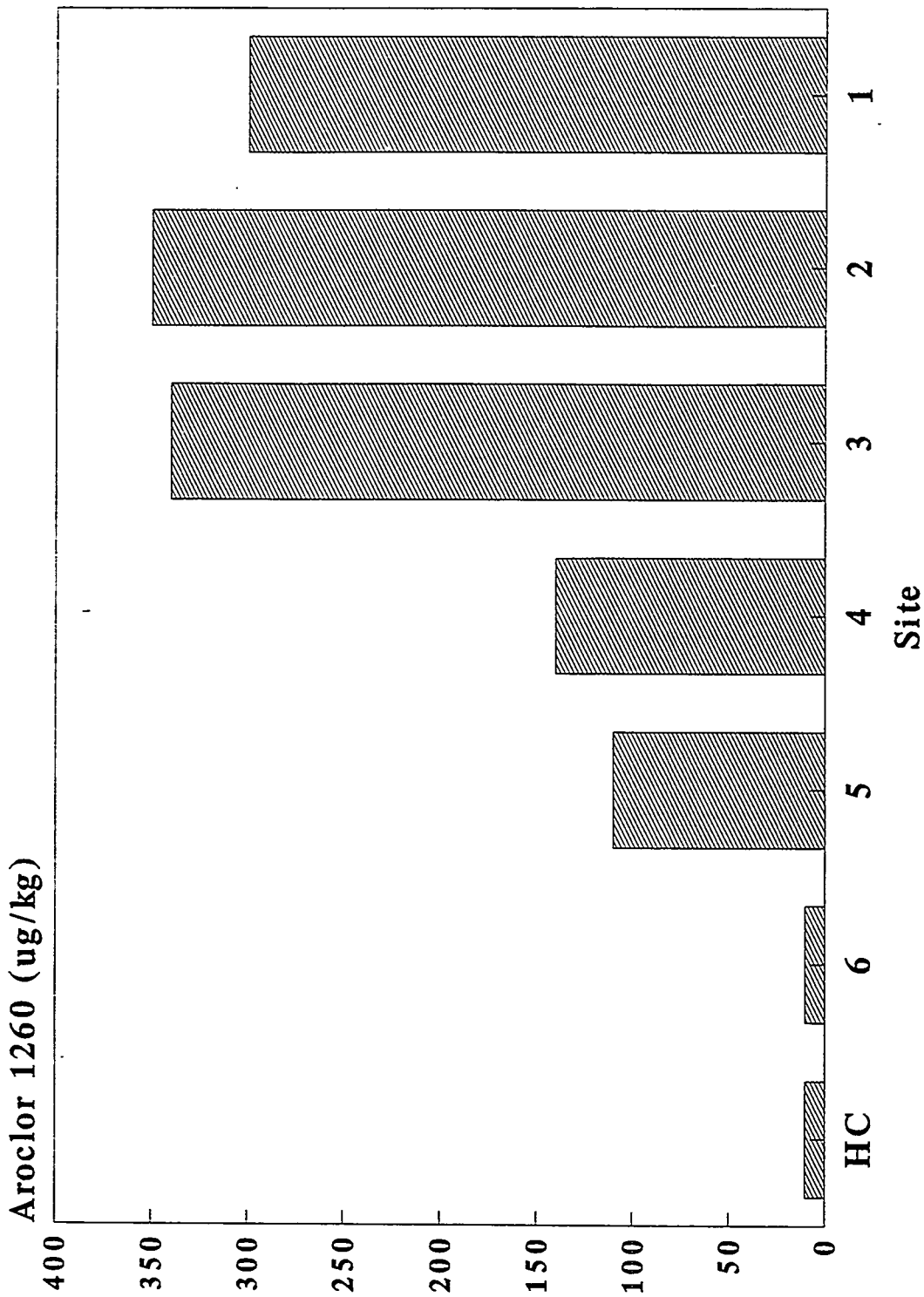

Fig. 6.53. Distribution of PCBs in terrestrial insects.

Table 6.33. Pesticides in terrestrial insects from EFPC and Hinds Creek[a]

Sample site	1	2	3	4	5	6	Reference
Aldrin	<6	8	7	8	<6	<6	4
Alpha-chlordane	<17	2	4	25	3	<17	<17
Alpha-BHC	<16	4	1	3	2	<16	3
Beta-BHC	<54	<54	<54	<54	6	<54	<54
DDD	<22	<22	<22	<22	<22	<22	<22
DDE	<28	51	<28	9	3	7	2
DDT	<14	<14	<14	<14	<14	<14	<14
Delta-BHC	<8	<8	<8	2	2	<8	2
Dieldrin	<39	<39	14	5	4	<39	1
Alpha-endosulfan	<14	<14	<14	<14	<14	<14	<14
Endosulfan	<17	<17	<17	<17	<17	<17	5
Endrin	<25	<25	<25	<25	<25	<25	<25
Endrin aldehyde	<51	<51	<51	<51	<51	4	<51
Endrin ketone	<16	<16	<16	<16	<16	1	0
Endosulfan sulfate	<19	<19	<19	<19	<19	<19	1
Gamma-BH	<15	2	3	3	5	2	3
Gamma-chlordane	<14	<14	<14	11	5	1	2
Heptachlor	<16	4	9	9	18	<16	8
Heptac epoxide	<9	8	7	22	8	4	4

Table 6.33. (continued)

Sample site	1	2	3	4	5	6	Reference
Methoxychlor	<150	<150	<150	<150	<150	<150	<150
Toxaphene	<150	<150	<150	<150	<150	<150	<150

[a]Concentrations in μg/kg

Table 6.34. PAHs in terrestrial insects from EFPC and Hinds Creek[a]

Sample site	1	2	3	4	5	6	Reference
Acenaphthylene	302	156	<380	<670	<645	<410	<1000
Acenaphtene	<655	<615	<280	<510	<480	<310	<750
Anthracene	27	25	11	20	56	11	26
Benzo(a)pyrene	2	2	1	2	3	1	2
Benzo(b)fluoranthene	<7	3	<3	<6	<5	<4	<9
Benzo(k)fluoranthene	1	2	1	1	1	1	2
Benzo(a)anthracene	<6	3	<3	<5	<4	<3	<7
Benzo(ghi)perylene	<20	<19	<9	<15	<15	<9	<23
Chrysene	<50	<47	<22	<39	<37	<23	<57
Dibenzo(ah)anthracene	<7	<7	<3	<6	<5	<4	<9
Fluoranthene	<83	25	<36	<65	<61	<39	<95
Fluorene	<92	28	<40	<73	<67	<43	<100
Indeno(123-cd)pyrene	<23	<21	0	<17	<17	<11	<26
Napthalene	208	4	7	199	<490	<320	12
Phenanthrene	<255	76	<110	<200	<190	<120	<290
Pyrene	<100	<94	<43	<79	<73	<47	<110

[a]Concentrations in μg/kg

Earthworms

Sampling methods and population survey results for earthworms are discussed in Sect. 6.3.3.8. Earthworms collected at six sites on EFPC and at the Mill Branch reference site were composited by site for whole-body analysis. No attempt was made to remove internal soil from the worms because the earthworm and gut contents were what would have been ingested by earthworm predators; therefore, measured body burdens may exceed actual levels. Analytical results indicate that earthworms were significantly exposed to contaminants in EFPC floodplain soil.

Mercury. Data for mercury in earthworm composites are presented in Table 6.35. Whole-body mercury concentrations in composites from the six sample sites in the EFPC floodplain ranged from 4.77 to 33.2 mg mercury/kg. Concentration at the Mill Branch reference site was less than the detection limit of 0.3 mg mercury/kg. The maximum concentration was observed at Site 2 (at over 100 times the concentration found in the reference sample), with a steady decrease downstream from that location (Fig. 6.54).

Other inorganics. The concentrations of inorganic contaminants in earthworm composites are given in Table 6.35. No composite from the EFPC floodplain sample sites had a higher level of antimony than the reference site. Arsenic, cadmium, selenium, uranium, and zinc in all composites were at concentrations above those of the reference sample, and chromium was less than in the reference sample only at Site 6. The maximum concentrations of arsenic and chromium in EFPC floodplain composites were less than twice the concentrations at the reference site. Cadmium was detected only at Sites 2 and 5. Zinc concentrations were lowest at Sites 1 and 6; concentrations at the other sites were approximately the same and were slightly more than twice the reference level. Selenium concentrations were highest at Sites 1 and 2; the maximum was more than two times the concentration in the reference sample. Uranium concentrations were highest at Sites 2 and 4 and lowest at Site 1; none was less than six times the concentration in the reference composite.

Although soil samples were not analyzed for inorganic contaminants other than mercury, the mercury levels can be used as indicators of deposition of contaminants from the Y-12 Plant. Concentrations of inorganic analytes in earthworm composites did not correlate well with the average concentration of mercury in the soil transects at the sample sites (Table 6.36).

PCBs, pesticides, and PAHs. The amount of tissue obtained during sampling of earthworms was not sufficient for analysis of organic contaminants.

Table 6.35. Inorganic analytes in earthworms homogenates from EFPC and Mill Branch[a]

Sample site	1	2	3	4	5	6	Reference
Mercury	6001	3213	28465	24605	19592	4772	<305
Antimony	34	56	360	42	35	34	375
Arsenic	1419	1944	1655	2063	1966	1349	1053
Cadmium	<1935	17098	<2389	<3343	119999	<2308	<1902
Chromium	8401	13644	10287	14644	10529	7776	8453
Selenium	5316	4515	2057	2816	3031	2814	1342
Uranium	3911	8672	6018	9127	5440	6128	1281
Zinc	67242	96257	101870	93143	107360	69187	51344

[a]Concentrations in μg/kg

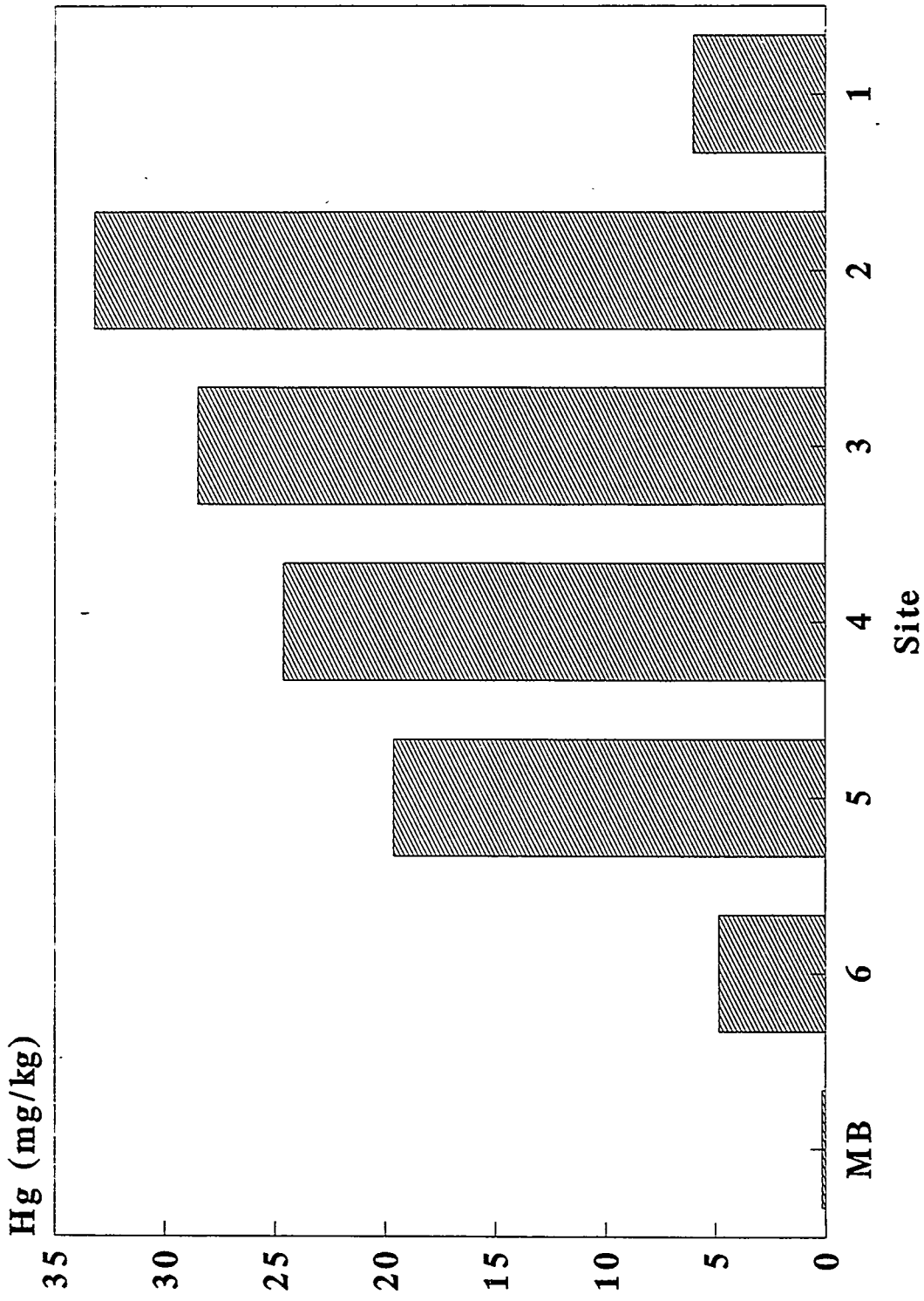

Fig. 6.54. Distribution of mercury in earthworms at EFPC and Mill Branch.

Table 6.36 Correlations of inorganic body burdens of earthworms with soil mercury concentrations at EFPC[a]

Correlations of inorganic burdens of earthworms with soil mercury concentration[a]	
Analyte	**Correlation coefficient[b]**
Mercury	0.11
Antimony	0.24
Arsenic	0.03
Chromium	0.0003
Uranium	0.018
Zinc	0.002

[a]Average mercury concentration in EFPC floodplain soil in the creek section in which samples were taken.
[b]Linear correlation coefficient for body burden vs. soil mercury concentration.

Plants

Grasses and vegetables were sampled for both the human risk and ecological risk assessments at three sites.

Mercury. Results of analyses for mercury in grasses and browse are presented in Table 6.37. Mercury levels associated with live grass were below detection limits at the reference site and at Sites 7 [100 m (330 ft) from creek edge] and 8 [100 m (330 ft) and creek edge locations]. The other samples ranged from 0.6 to 1.0 mg mercury/kg. Mercury associated with dead grass was below detection limits in the reference sample and at Site 7 [100 m (330 ft) from creek edge]. In four samples of dead grass the range was 0.19 to 0.84 mg mercury/kg, but at Site 3 (along the creek edge) the reported value was ~17 mg mercury/kg. The average mercury concentration in all EFPC floodplain samples was ~1.5 mg/kg. In general (i.e., in four samples out of five), grasses near the creek showed higher levels of mercury than those at the 100-m (330-ft) location. Dead grass was expected to be more contaminated with adherent sediment (Van Winkle et al. 1984), but that was true in only three of five locations.

Mercury was not detected in browse from the reference site, but was reported at 0.1 mg/kg at 100 m (330 ft) and at 0.65 mg/kg at the creek edge at Site 3.

Mercury levels in garden vegetables ranged from below detection levels at 0.03 mg/kg in tomato fruits to 3.2 mg/kg in kale leaves (Table 6.38). It is likely that adherent soil contributed to the observed concentrations in kale leaves and in kale and tomato roots. Beets, which were washed thoroughly before being analyzed, had much lower mercury concentrations.

Organics. Organic analytes were not measured in plant samples. Uptake of PCBs and PAHs by plants is considered to be insignificant (Travis and Arms 1988).

6.2.3.3 Comparison of historical and current exposures

This subsection compares body burden concentrations of contaminants in biota collected from EFPC during this investigation and those reported in historical studies conducted during May 1982 through November 1989. The comparisons are arranged by taxa and contaminant. When comparing the contaminant body burdens in fish from this investigation with those from historical studies, two important qualifiers should be kept in mind: (1) contaminant concentrations in fish from this study are for whole-body composited samples, whereas the historical studies describe data for fish fillets and (2) mercury analyses of the fish were conducted using alternate analytical methodologies for this investigation (neutron activation analysis) compared with those

Table 6.37. Mercury concentrations in grass and browse samples at EFPC and Hinds Creek (mg/kg)

Site	Location	Sample type		
		Live grass	Dead grass	Browse
Site 3	creek edge	0.60	16.98	0.65
	100 m (330 ft) from creek edge	0.83	0.19	0.10
Site 7	creek edge	1.00	0.46	–
	100 m (330 ft) from creek edge	<0.09	<0.19	–
Site 8	creek edge	<0.09	0.84	–
	100 m (330 ft) from creek edge	<0.06	0.48	–
	garden plot	0.26	–	–
Hinds Creek	creek edge	<0.09	<0.21	<0.03

Table 6.38. Mercury in garden crops at EFPC and reference site (mg/kg)
(summary of preliminary results, 10/28/92)

Sample type	LOCATION		
	Site 3 near creek	Site 3 100 m (330 ft)	Reference
Beet root	--	2.72	--
	--	1.08	--
	--	0.63	--
	--	0.76	--
Kale root[a]	72.6	240.2	<0.18
	48.3	88.5	<0.25
	59.0	243.5	<0.53
Kale leaf	3.20	0.18	<0.05
	0.35	0.13	<0.04
	0.17	1.28	<0.04
	0.31	--	--
Tomato root[a]	134.3	258.0	0.18
	61.0	102.5	<0.10
	152.3	33.5	<0.53
Tomato fruit	<0.04	<0.056	<0.02
	<0.03	0.42	--
	<0.03	--	--

[a]Roots were not washed free of all adherent soil, so the reported values do not reflect uptake of soil contaminants into roots.

of the historical studies (mercury extraction followed by cold-vapor atomic absorption spectroscopy). The relationship of concentrations in whole body to fillets is discussed in Sect. 6.1.5.4 under PARCC parameters.

Fish

Mercury. Table 6.39 summarizes the total mercury concentrations in redbreast sunfish, bluegill, and stonerollers collected from various sample sites in EFPC during studies conducted from May 1982 through October 1991. The studies include this investigation, BMAP studies (Loar 1992; Hinzman 1992), the TVA study (TVA 1985), and the study by Van Winkle et al. (1984). Bluegill are included in this discussion because they (1) belong to the same genus as redbreast sunfish (*Lepomis*), (2) have the same trophic classification (insectivore), and (3) were collected during many years at sampling locations near the ones used during this study in EFPC.

During the 1982 study performed by Van Winkle et al., maximum mercury concentrations of 2.13 mg/kg were obtained in bluegill collected from the EFPC sampling site nearest the Y-12 Plant (EFK 22.8), and decreased concentrations were obtained downstream from the plant. In contrast, the greatest mercury concentrations in redbreast sunfish in this study (1.86 mg/kg) were not observed closest to the Y-12 Plant, but occurred in samples from Site 3, ~6.4 km downstream from Lake Reality. Mercury concentrations in redbreast sunfish did progressively decrease downstream from Sites 3 to 6. Furthermore, whole body mercury concentrations in redbreast sunfish collected from Sites 1, 4, and 6 in this study were approximately one-half to one-third less than the mercury concentrations in bluegill collected from the same areas during May 1982 study (Table 6.39). The probable explanation for the lower mercury concentrations in these redbreast sunfish is that two remedial actions were performed at the Y-12 Plant during May 1985 through November 1988, and these actions reduced the amount of mercury transported into EFPC. The remedial actions (completed during 1986 through 1987) included isolating, cleaning, and relining the storm and process water drains that contributed the most mercury to EFPC, and rerouting EFPC around New Hope Pond and through Lake Reality (Hinzman 1992). In this investigation and in the 1982 study, mercury concentrations in redbreast sunfish from all sampling sites in EFPC exceeded the mercury concentrations in redbreast sunfish samples collected from a reference site.

In the TVA instream contaminant study of 1984 (TVA 1985), as was the case during the previously discussed 1982 study (Van Winkle et al. 1984), the maximum mercury concentration in redbreast sunfish (1.7 mg/kg) was obtained in the sample collected nearest the Y-12 Plant (EFK 22.2), and the concentrations decreased in samples collected downstream from the plant

Table 6.39. Summary of total mercury concentrations in fish collected from EFPC, May 1982–October 1991[a]

Study	Sampling location										
	EFK 2.1-4.0	EFK 4.1-6.0	EFK 6.1-8.0	EFK 8.1-10.0	EFK 10.1-12.0	EFK 12.1-14.0	EFK 14.1-16.0	EFK 16.1-18.0	EFK 18.1-20.0	EFK 20.1-22.0	EFK 22.1-24.0
SAIC (10/91)	0.18		0.48		0.86			1.86		1.08	1.08
• Redbreast (whole)											
• Stonerollers (whole)	0.36		0.72		0.71			1.93		6.37	3.14
BMAP (12/89 through 5/85)											
• Redbreast (12/89)[b]	0.64		0.74			0.88			0.96		1.04
(5/89)[b]	0.46		0.47			0.69			0.65		1.01
(12/88)[b]	0.39		0.67			0.76			0.76		1.36
(5/88)[b]	0.35		0.60			0.66			0.95		1.45
(12/87)[b]	0.43		0.75			1.03			1.25		1.57
(5/87)[b]	0.47		0.82			0.83			1.15		1.49
(12/86)[b]	0.40		0.70			0.91			0.91		1.70
(5/86)[b]	0.34		0.41			0.59			0.87		1.19
(12/85)[b]	0.36		0.56			0.73			0.83		1.26
(5/85)[b]	0.45		0.38			0.65			0.77		0.62
• Bluegill (12/89)	0.44		NC[c]			NC			NC		0.84
(5/89)	0.22		NC			NC			NC		0.51
(12/88)	0.39		NC			NC			NC		0.80
(5/88)	0.32		NC			NC			NC		1.08
(12/87)	0.43		NC			NC			NC		1.76
(5/87)	0.38		0.60			NC			NC		0.70
(12/86)	0.38		0.55			0.31			NC		1.16
(5/86)	0.29		NC			0.47			0.40		0.74
(12/85)	0.38		0.40			NC			0.96		0.86
(5/85)	0.48		0.65			0.37			NC		0.83
TVA (5/84)							0.96				1.7
• Redbreast			0.67				0.80				0.68
• Bluegill			0.89								

Table 6.39. (continued)

Study	Sampling location										
	EFK 2.1-4.0	EFK 4.1-6.0	EFK 6.1-8.0	EFK 8.1-10.0	EFK 10.1-12.0	EFK 12.1-14.0	EFK 14.1-16.0	EFK 16.1-18.0	EFK 18.1-20.0	EFK 20.1-22.0	EFK 22.1-24.0
Van Winkle et al. (5/82) • Bluegill[b]	0.66					1.45					2.13 1.66

[a]Mercury is measured in mg/kg.
[b]Fillets.
[c]NC = fish not collected.

(Table 6.39). In contrast, the maximum mercury concentrations in redbreast sunfish from this investigation (1.86 mg/kg) were observed in samples from Site 3 (EFK 17.6). Mercury concentrations in redbreast sunfish from Sites 1 and 5 in this study were ~30 to 40% less than the mercury concentrations in redbreast collected near the same locations during the TVA instream contaminant study in May 1984 (Table 6.39). However, redbreast sunfish collected from Site 3 in this study contained twice as much mercury (1.86 versus 0.96 mg mercury/kg) as the redbreast sunfish collected ~3 km (1.8 mi) downstream during the May 1984 study. The disparities between mercury concentrations in redbreast sunfish collected from this study and those from the TVA investigation are most likely attributable to the two previously described remedial actions on the Y-12 Plant site. In both studies, mercury concentrations in redbreast sunfish from all sampling sites in EFPC exceeded the mercury concentrations in redbreast sunfish samples collected from a reference site.

In the BMAP studies from May 1985 through December 1989 (Loar 1992; Hinzman 1992), the maximum mercury concentrations in redbreast sunfish were generally obtained from the sample collected closest to the Y-12 Plant (EFK 23.4), and the concentrations decreased in samples collected downstream from the plant (Table 6.39). In this study, the mercury concentrations in redbreast sunfish collected from the site closest to the Y-12 Plant (EFK 22.2) were ~15 to 40% lower than the mean concentrations in redbreast sunfish collected during the December 1985 through December 1988 BMAP sampling. However, mercury concentrations in the redbreast sunfish collected at EFK 23.4 during the May 1989 through December 1989 BMAP sampling (at 1.01 and 1.04 mg/kg) were nearly identical to the concentrations observed in this study (at 1.08 mg/kg), suggesting that the mercury levels in redbreast sunfish near the Y-12 Plant may be declining in relation to the historical levels of the early and mid-1980s.

During the BMAP studies from May 1985 through December 1989, the mean mercury concentrations in redbreast sunfish collected from EFK 18.2 ranged from 0.65 to 1.25 mg/kg, whereas the concentration in redbreast sunfish from Site 3 (EFK 17.6) was ~33 to 65% higher in this study than were any of the BMAP EFK 18.2 sampling means. As discussed in Sect. 6.3.2.3, the higher mercury concentrations observed in insects at this site might explain the higher mercury concentrations in sunfish, which feed on insects. The mercury concentrations in redbreast sunfish collected from Sites 4 (EFK 10.8) and 5 (EFK 7.3) in this study (at 0.86 and 0.48 mg/kg, respectively) were similar to the mean mercury concentrations in redbreast sunfish (at 0.66 to 1.03 mg/kg and 0.38 to 0.82 mg/kg, respectively) collected from the closest comparable BMAP sites (EFK 13.8 and EFK 6.3, respectively). At the site farthest downstream from the Y-12 Plant, the mercury levels in redbreast sunfish (at 0.18 mg/kg at EFK 2.3) were

approximately ~50 to 72% lower in this study than were the mercury levels in redbreast sunfish collected from the BMAP site at EFK 2.1. In the BMAP studies and in this investigation, the mercury concentrations in all redbreast sunfish samples from EFPC exceeded the mercury levels in redbreast collected from the reference site.

Bluegill were captured in sufficient abundance for mercury analyses at EFK 23.4 and EFK 2.1 during all BMAP sampling periods from May 1985 through December 1989, but were only occasionally captured at the other three sites (Table 6.39). The mean mercury concentrations in bluegill collected from EFK 23.4 were nearly always less than the concentrations in redbreast sunfish, except during the May 1985 and December 1987 sampling periods (Table 6.39). The lower mercury concentrations in bluegill are most likely due to interspecies physiological differences between redbreast and bluegill. Mercury concentrations in bluegill and redbreast collected from EFK 2.1 during the BMAP studies were similar.

PCBs. Table 6.40 summarizes the total PCB or Aroclor 1260 concentrations in redbreast sunfish, bluegill, and carp that were collected from various sample sites in EFPC during studies conducted from May 1985 through October 1991. These studies include this investigation and the Y-12 Plant BMAP studies (Loar 1992; Hinzman 1992). Bluegill are included in this discussion for the reasons stated in the previous subsection on mercury comparisons in fish, and carp are included for interspecies comparison.

All fish results exhibit an overall trend of decreasing PCB concentration from the headwaters to the confluence with Poplar Creek. The strongest decreasing trend was observed in redbreast sunfish; carp were less consistent in exhibiting a decreasing trend in the downstream direction. PCB concentrations in whole redbreast sunfish were consistently higher than in redbreast fillets. Concentrations in whole bluegill were comparable to redbreast fillets. Whole stoneroller concentrations were more than three times as high as whole redbreast concentrations at the two farthest upstream sites, whereas redbreast concentrations were twice as high as stoneroller concentrations in the middle reaches of EFPC.

Throughout the length of the stream, PCB concentrations in redbreast fillets ranged from 0.18 to 1.74 mg/kg. Bluegills ranged from 0.05 to 1.45 mg/kg PCB. Carp ranged from 0.17 to 3.25 mg/kg PCB. Aroclor 1260 concentrations ranged from 0.47 to 2.80 mg/kg in whole redbreast and from 0.82 to 8.10 mg/kg in whole stonerollers.

Table 6.40. Summary of total PCB or Aroclor 1260 concentrations in fish collected from EFPC, May 1982–October 1991[a]

Study	Sampling location										
	EFK 2.1-4.0	EFK 4.1-6.0	EFK 6.1-8.0	EFK 8.1-10.0	EFK 10.1-12.0	EFK 12.1-14.0	EFK 14.1-16.0	EFK 16.1-18.0	EFK 18.1-20.0	EFK 20.1-22.0	EFK 22.1-24.0
SAIC (10/91)											
• Redbreast (whole, Aroclor 1260)	0.47		0.99		1.60			2.80		2.20	2.50
• Stonerollers (whole, Aroclor 1260)	NC[b]		1.10		0.82			1.90		7.35	8.10
BMAP (12/89 through 5/85)											
• Redbreast (12/89)[c]											
(5/89)[c]											
(12/88)[c]											
(5/88)[c]	0.18		0.19			0.36			0.43		0.86
(12/87)[c]	0.14		0.18			0.62			0.76		0.61
(5/87)[c]	0.15		0.18			0.30			0.57		1.04
(12/86)[c]	0.08		0.10			0.31			0.28		1.74
(5/86)[c]	0.22		0.16			0.26			0.21		0.69
(12/85)[c]	0.26		0.21			0.22			0.27		0.49
(5/85)[c]	0.19		0.25			0.39			0.34		0.48
• Bluegill (5/88)[c]	0.27		NC			NC			NC		.66
(12/87)[c]	0.14		0.31			NC			NC		1.45
(5/87)[c]	0.17		0.13			NC			NC		0.46
(12/86)[c]	0.12		0.05			NC			NC		0.70
(5/86)[c]	0.31		0.20			NC			NC		0.26
(12/85)[c]	0.20		0.24			NC			0.75		0.36
(5/85)[c]	0.18		NC			0.16			0.07		0.34

Table 6.40. (continued)

Study		Sampling location										
		EFK 2.1-4.0	EFK 4.1-6.0	EFK 6.1-8.0	EFK 8.1-10.0	EFK 10.1-12.0	EFK 12.1-14.0	EFK 14.1-16.0	EFK 16.1-18.0	EFK 18.1-20.0	EFK 20.1-22.0	EFK 22.1-24.0
• Carp	(5/88)[c]	0.28		0.51	NC		1.52			1.11		NC
	(12/87)[c]	0.17		0.62	NC		NC			2.03		NC
	(5/87)[c]	1.11		0.82	NC		0.96			0.97		0.59
	(12/86)[c]	NC		1.22	0.52		NC			NC		NC
	(5/86)[c]	0.44		0.44			0.75			0.83		NC
	(12/85)[c]	NC		1.29			1.95			NC		NC
	(5/85)[c]	3.25		1.70			2.94			NC		2.52

[a]PCBs and Aroclor 1260 concentrations are measured in mg/kg.
[b]NC = fish not collected.
[c]Fillets.

Crayfish

Mercury. In the TVA instream contaminant study of 1984 (TVA 1985), mercury concentrations in crayfish collected from three sites in EFPC (EFK 22.2, EFK 14.2, and EFK 6.4) were 0.82, 0.62, and 0.29 mg/kg, respectively, indicating a decrease in concentrations downstream from the Y-12 Plant. In this investigation, the mercury concentration in crayfish collected from Site 1 (EFK 22.2) was 6.02 mg/kg, nearly an order of magnitude greater than that observed during the TVA study. Unfortunately, no crayfish were captured during this study at the sites located nearest the other two TVA sampling sites. In both studies, the mercury concentrations in crayfish decreased downstream from the Y-12 Plant.

6.3 EFFECTS ASSESSMENT

According to the *Framework for Ecological Risk Assessment* (EPA 1992a), the ERA process consists of four steps—problem formulation, exposure characterization, effects characterization and risk characterization. Section 6.3 discusses ecological effects characterization, which evaluates the response to chemical and physical stressors in terms of the selected assessment and measurement endpoints and, depending on the parameters of exposure, results in a profile of response to stressor at concentrations or doses or other units of stress to which indicator populations and habitats are exposed. The four steps of the ERA process are described in more detail in Sect. 6.1.2.

6.3.1 Conventional Toxicity Data

6.3.1.1 Toxicity reference data

Data on the toxicity of organic and inorganic mercury species to freshwater fish, as reviewed by Mance (1987), Eisler (1987), and Call et al. (1983), are presented in Table 6.41. The dose-effect relationship for both organic and inorganic mercury species is plotted in Fig. 6.55. Although Snarski and Olson (1982) reported no effects on juvenile fathead minnows (*Pimephales promelas*) exposed to 0.00026 to 0.00058 mg inorganic mercury/L, significant reduction in length and weight of fathead minnows at 29-30-d post-hatch was observed at 0.00023 mg inorganic mercury/L (Call et al. 1983). Mortality was reported from 0.00293 to 4.1 mg/L. Effects such as reduced weight and growth, reproductive problems, and spasms and deformities occurred at intermediate but overlapping concentrations between the levels of no effect and of mortality.

Table 6.41. Mercury dose-effect data for freshwater fish

Effect	Fish species	Stage	Mercury species	Concentration mg/L	Duration (days)	Reference
Inorganics						
No effect	*Pimphales promelas*	J[a]	$HgCl_2$ (*Artemia* diet)	0.00026	30	Snarski & Olson 1982
No effect	*P. promelas*	J	Artificial diet	0.00031	60	Snarski & Olson 1982
No effects on growth or mortality	*P. promelas*	J	Artificial diet	0.00058	30	Snarski & Olson 1982
Spawning unaffected	*P. promelas*	LC[b]	$HgCl_2$ (*Artemia* diet)	0.0005	287	Snarski & Olson 1982
Significant reduction in weight and length of fish 36-d post hatch	*P. promelas*	EL	$HgCl_2$	0.00023	29-30	Call et al. 1983
Growth reduced	*P. promelas*	J	$HgCl_2$ (*Artemia* diet)	0.0005	30	Snarski & Olson 1982
Growth reduced	*P. promelas*	J	Artificial diet	0.00058	60	Snarski & Olson 1982
Growth reduced	*P. promelas*	J	Artificial diet	0.00127	30	Snarski & Olson 1982
Growth reduced	*P. promelas*	J	$HgCl_2$ (*Artemia* diet)	0.00201	60	Snarski & Olson 1982
Reproductive effects-spawning prevented	*P. promelas*	LC	$HgCl_2$ (*Artemia* diet)	0.00102	287	Snarski & Olson 1982
Significant increase in abnormal fry with gross morphological anomolies	*P. promelas*	EL	$HgCl_2$	0.00185		Call et al. 1983
Reproductive effects-suppression of sexual maturation	*P. promelas*	LC	$HgCl_2$ (*Artemia* diet)	0.00369	287	Snarski & Olson 1982
Reduced weight ♀	*P. promelas*	LC	$HgCl_2$ (*Artemia* diet)	0.00102	287	Snarski & Olson 1982
Reduced weight ♂	*P. promelas*	LC	$HgCl_2$ (*Artemia* diet)	0.00367	287	Snarski & Olson 1982

Table 6.41. (continued)

Effect	Fish species	Stage	Mercury species	Concentration mg/L	Duration (days)	Reference
26% spinal deformity	*P. promelas*	J	Artificial diet	0.00451	60	Snarski & Olson 1982
50% mortality	*P. promelas*	J	Artificial diet	0.00451	30, 60	Snarski & Olson 1982
Significant reduction in survival at 29-d post hatch	*P. promelas*	EL	$HgCl_2$	0.00185	29	Call et al. 1983
EC_{50} (death and deformity)	*Carassius auratus*	EL	$HgCl_2$	0.0007	7	Birge 1978; Birge et al. 1979
EC_{50} (death and deformity	*Ictalurus punctatus*	EL	$HgCl_2$	0.0003	10	Birge et al. 1979
5-10% mortality	*Channa punctatus*	55 g	$HgCl_2$	0.3	15	Sastry & Sharma 1980
10-20% mortality	*C. punctatus*	55 g	$HgCl_2$	0.3	30	Sastry & Sharma 1980
LC_{50}[e]	*Salmo gairdneri*	2 m	mercurous nitrate	0.033	4	Hale 1977
LC_{50}	*P. promelas*	3.2-4.2 cm	mercuric acetate	0.064	4	Curtis et al. 1979
LC_{50}	*P. promelas*	J	$HgCl_2$	0.084	6	Snarski & Olson 1982
LC_{50}	*Roccus saxatilis*	A[c]	Hg^{2+}	0.09	4	Rehwoldt et al. 1972
LC_{50}	*P. promelas*	3.2-4.2 cm	$HgCl_2$	0.074	7	Snarski & Olson 1982
LC_{50}	*Fundulus diaphanus*	A	Hg^{2+}	0.11	4	Rehwoldt et al. 1972
LC_{50}	*F. diaphanus*	A	Hg^{2+}	0.16	2	Rehwoldt et al. 1972
LC_{50}	*R. saxatilis*	A	Hg^{2+}	0.14	2	Rehwoldt et al. 1972
LC_{50}	*Anguilla rostrata*	A	Hg^{2+}	0.14	4	Rehwoldt et al. 1972
LC_{50}	*A. rostrata*	A	Hg^{2+}	0.19	2	Rehwoldt et al. 1972
LC_{50}	*A. promelas*	3.2-4.2 cm	mercuric thiocyanate	0.181	4	Curtis et al. 1979
LC_{50}	*P. promelas*	3.2-4.2 cm	mercuric acetate	0.19	4	Curtis et al. 1979
LC_{50}	*P. promelas*	J	$HgCl_2$	0.112	5	Snarski & Olson 1982

Table 6.41. (continued)

Effect	Fish species	Stage	Mercury species	Concentration mg/L	Duration (days)	Reference
LC_{50}	*P. promelas*	J	$HgCl_2$	0.168	4	Snarski & Olson 1982
LC_{50}	*Lepomis macrochirus*	0.6 g	$HgCl_2$	0.16	3, 4	Holcombe et al. 1983
LC_{50}	*P. promelas*	3.2-4.2 cm	mercuric thiocyamate	0.15	4	Curtis et al. 1979
LC_{50}	*L. macrochirus*	0.6g	$HgCl_2$	0.25	2	Holcombe et al. 1983
LC_{50}	*L. macrochirus*	0.6g	$HgCl_2$	0.28	1	Holcombe et al. 1983
LC_{50}	*Cyprinus carpio*	A	Hg^{2+}	0.18	4	Rehwoldt et al. 1972
LC_{50}	*C. carpio*	A	Hg^{2+}	0.21	2	Rehwoldt et al. 1972
LC_{50}	*Roccus americanus*	A	Hg^{2+}	0.22	4	Rehwoldt et al. 1972
LC_{50}	*R. saxatilis*	A	Hg^{2+}	0.22	1	Rehwoldt et al. 1972
LC_{50}	*A. rostrata*	A	Hg^{2+}	0.25	1	Rehwoldt et al. 1972
LC_{50}	*L. gibbosus*	A	Hg^{2+}	0.30	4	Rehwoldt et al. 1972
LC_{50}	*R. americanus*	A	Hg^{2+}	0.34	2	Rehwoldt et al. 1972
LC_{50}	*Cyprinus carpio*	A	Hg^{2+}	0.33	1	Rehwoldt et al. 1972
LC_{50}	*Carassius auratus*	A	$HgCl_2$	0.35	2	Heisinger et al. 1979
LC_{50}	*L. gibbosus*	A	Hg^{2+}	0.39	2	Rehwoldt et al. 1972
LC_{50}	*L. gibbosus*	A	Hg^{2+}	0.41	1	Rehwoldt et al. 1972
LC_{50}	*R. americanus*	A	Hg^{2+}	0.42	1	Rehwoldt et al. 1972
LC_{50}	*F. diaphanus*	A	Hg^{2+}	0.27	1	Rehwoldt et al. 1972
LC_{50}	*P. promelas*	3.2-4.2 cm	mercuric thiocyanate	0.39	1, 2	Curtis et al. 1979
LC_{50}	*P. promelas*	3.2-4.2 cm	mercuric thiocyanate	0.47	2	Curtis et al. 1979

Table 6.41. (continued)

Effect	Fish species	Stage	Mercury species	Concentration mg/L	Duration (days)	Reference
LC_{50}	*P. promelas*	3.2-4.2 cm	mercuric acetate	0.53	1	Holcombe et al. 1983
LC_{50}	*Catostomus commersoni*	A	HgCl	0.740	3	Duncan & Klaverkamp 1983
LC_{50}	*C. commersoni*	A	HgCl	0.830	2	Duncan & Klaverkamp 1983
LC_{50}	*C. commersoni*	A	$HgCl_2$	0.687	4	Duncan & Klaverkamp 1983
LC_{50}	*Tilapia mossambia*	A	$HgCl_2$	1.0	4	Qureshi & Saksena 1980
LC_{50}	*T. mossambia*	A	$HgCl_2$	1.4	2	Qureshi & Saksena 1980
LC_{50}	*T. mossambia*	A	$HgCl_2$	1.8	1	Qureshi & Saksena 1980
LC_{50}	*C. commersoni*	A	HgCl	1.6	0.5, 1	Duncan & Klaverkamp 1983
LC_{50}	*Channa gachna*	A	$HgCl_2$	1.4	4	Hanumante & Kulkarni 1979
LC_{50}	*C. gachna*	A	$HgCl_2$	1.6	3	Hanumante & Kulkarni 1979
LC_{50}	*C. gachna*	A	$HgCl_2$	2.2	2	Hanumante & Kulkarni 1979
LC_{50}	*C. punctatus*	A	$HgCl_2$	1.8	4	Sastry & Sharma 1980
LC_{50}	*C. gachna*	A	$HgCl_2$	4.1	1	
Organic						

Table 6.41. (continued)

Effect	Fish species	Stage	Mercury species	Concentration mg/L	Duration (days)	Reference
No adverse effect	*Salvelinus fontinalis*	Y-A[d]	methyl mercuric chloride	0.00093	271	McKim et al. 1976
Reproductive: 100% spawning mortality	*S. fontinalis*	A	methyl mercuric chloride	0.00091	730	McKim et al. 1976
Reproductive: no spawning	*S. fontinalis*	Y-A	methyl mercuric chloride	0.00293	272	McKim et al. 1976
Spasms & deformity	*S. fontinalis*	Y-A	methyl mercuric chloride	0.0093	140	McKim et al. 1976
88% mortality	*S. fontinalis*	Y-A	methyl mercuric chloride	0.00293	272	McKim et al. 1976
LC_{50}	*S. fontinalis*	J	methyl mercuric chloride	0.065	4	McKim et al. 1976
LC_{50}	*S. fontinalis*	J	methyl mercuric chloride	0.084	4	McKim et al. 1976

[a]J = juvenile
[b]LC = lifecycle
[c]A = adult
[d]Y = yearling
[e]LC^{50} = lethal concentration fifty (the concentration that kills 50% of test organisms)

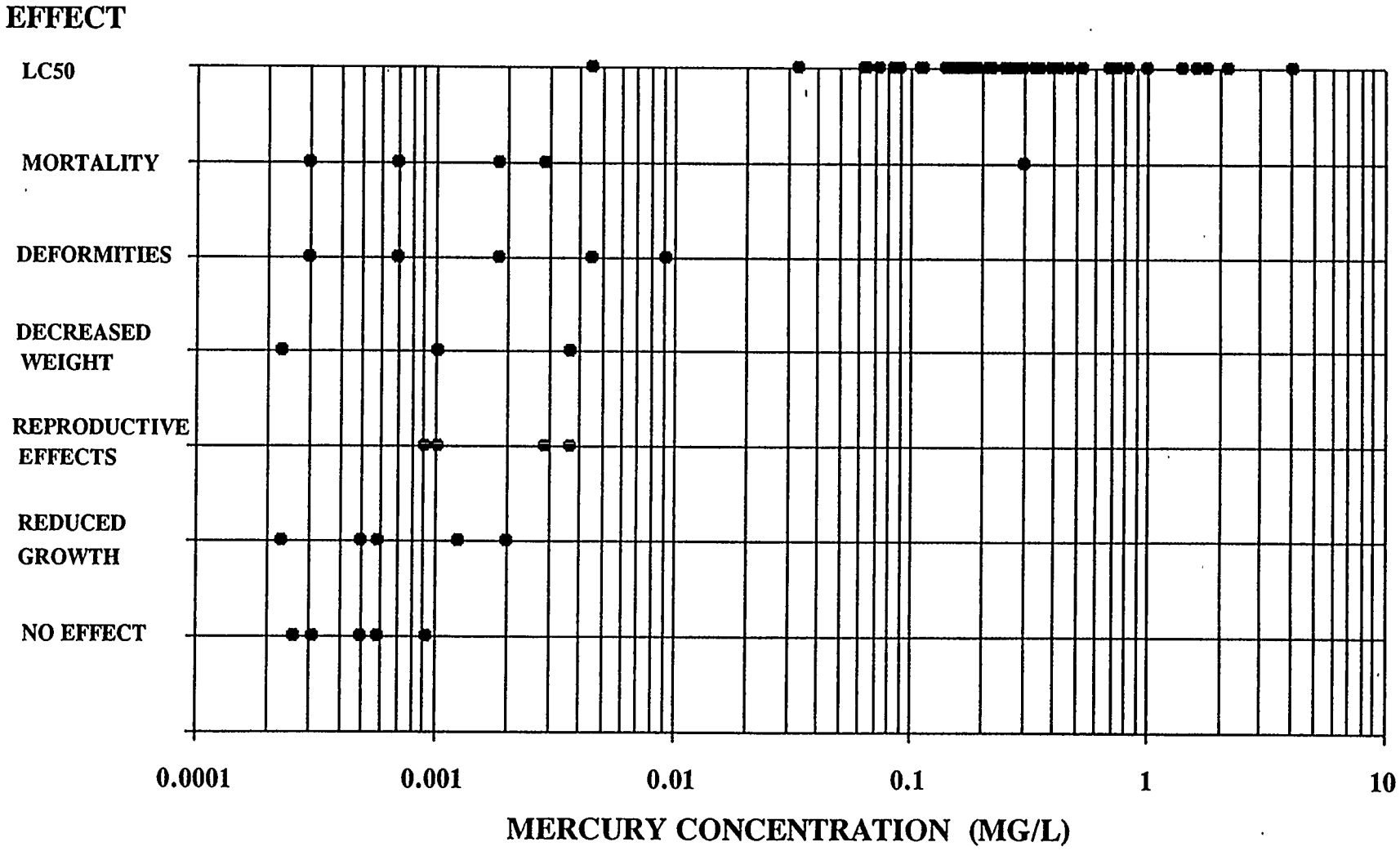

Fig. 6.55. Dose-effect pattern for mercury species and freshwater organisms.

Snarski and Olson (1982) reported on the effects of mercuric chloride ($HgCl_2$) to the fathead minnow. No effects were observed at 0.00026 mg/L, and reduced growth and weight are reported in the range of 0.0005 to 0.003 mg/L. Although there were no reproductive effects observed at 0.0005 in the life cycle test (287 d), spawning ceased at 0.00102 mg/L. Similarly, Snarski and Olson (1982) report that with very low levels of mercury in an artificial diet, there are no effects on this fish species. There were no effects from 0.00058 mg/L for 30 d, but after 60 d, growth was found to be reduced. An artificial diet, coupled with 0.00451 mg/L $HgCl_2$ exposures for 30 or 60 d produced 50% mortality, and during the longer period of exposure, juveniles also developed spinal deformities. The LC_{50} measurements in Table 6.41 reflect lethal effects for shorter exposure times (0.5 to 7 d); the lowest was 0.033 mg/L for mercurous nitrate in rainbow trout *(Salmo gairdneri)*.

Table 6.42 presents data on the effects of ingestion of inorganic mercury by mammals. A plot of these data (Fig. 6.56) reveals the variability of mercury toxicity to mammals, which is partially confounded due to differences in interspecies variability, mercury speciation, and exposure duration. For example, Table 6.42 gives no data on the no-effects level for the rat. Weight changes, histological damage to the brain and kidneys, and symptoms of mercurialism (stomatitis, gum and teeth problems, erethism, salivation, tremors, and uncoordination) were reported in rats ingesting concentrations of 100 to >200 ppm of inorganic mercury for periods from 10 months to 1 year. Acute lethal effects (LD_{50} measurements) appeared with ingestion of as little as 1 mg/kg of mercuric chloride.

Cadmium

Cadmium (CAS No. 7440-43-9) is a soft, bluish white lustrous metal with a natural abundance of 0.1 to 0.2 mg/kg in the earth's crust. Cadmium is relatively mobile in the aquatic environment compared to other heavy metals. Complexation with organic materials appears to be the mechanism of removal of cadmium from aqueous media. The mobility of cadmium transport in the soil is not clearly known; however, cadmium uptake by plants is considered significant. Cadmium accumulates in the tissues of both freshwater and marine organisms by factors of hundreds to thousands greater than that of the concentration in surrounding water. The total body burden is typically correlated with duration of exposure.

Approximately 6 to 10% of ingested cadmium is absorbed by experimental animals. Cadmium is transported largely in the red blood cells. Selective accumulation of cadmium in the renal cortex has been reported, although in cases of excessive exposure, the liver may contain more. During inhalation exposure, most cadmium salts have short-term retention in the lungs.

Table 6.42. Mercury dose-effect data for mammals[a]

Effect	Organism	Inorganic mercury-species	Concentration	Duration	Reference
No clinical effect	Mink	mercuric chloride	10 mg/kg/d[b]	29 d	Aulerich et al. 1974
Weight changes	Rat	mercuric acetate	160 ppm	1 year	Fitzhugh et al. 1950
Histological damage to brain and kidneys	Rat	$Hg\ Cl_2$	100-200 mg/kg/d[b]	10 month	Enders & Noetzel 1965
Histological damage to kidney	Rat	mercuric acetate	160 ppm	1 year	Fitzhugh et al. 1950
Symptoms of mercurialism	Rat	calomel (mercurous chloride)	210 mg/kg		Lehman 1951
MLD	Rat	mercurous cyanide	25 mg/kg		Cassidy & Farr 1978
Lethal[c]	Dog	mercuric chloride	60 mg/kg		Cassidy & Farr 1978
Lethal[c]	Dog	mercuric chloride	160 mg/kg		Cassidy & Farr 1978
LD_{50}[d]	Rat	HgCl	1 mg/kg		NIOSH 1983
LD_{50}	Mouse	$Hg(NO_3)_2$	8 mg/kg		NIOSH 1983
LD_{50}	Rat	$HgCl_2$	37 mg/kg		Lehman 1951
LD_{50}	Rat	mercuric iodide	40 mg/kg		Cassidy & Furr 1978
LD_{50}	Mouse	mercuric iodide	110 mg/kg		Cassidy & Furr 1978
LD_{50}	Rat	mercurous chloride	210 mg/kg		Cassidy & Furr 1978
LD_{50}	Rat	HgCl	210 mg/kg		NIOSH 1983
LD_{50}	Mouse	$HgNO_3$	388 mg/kg		NIOSH 1983
100% Fatal in ~ 2 months	Mink	-	1.0 mg/kg	up to 2 months	Sheffy & St. Amant 1982
Elevated residues in kidney and liver Death	Mink	-	5.0 mg/kg	up to 30-37 days	Sheffy & St. Amant 1982

Table 6.42. (continued)

Effect	Organism	Inorganic mercury-species	Concentration	Duration	Reference
LD_{50}	Mule deer	-	17.88 mg/kg bw	single dose	Hudson et al. 1984

[a]Oral route.
[b]d = days.
[c]100% of test animals died.
[d]LD_{50} = lethal dose fifty (dose at which 50% of test organisms are killed).

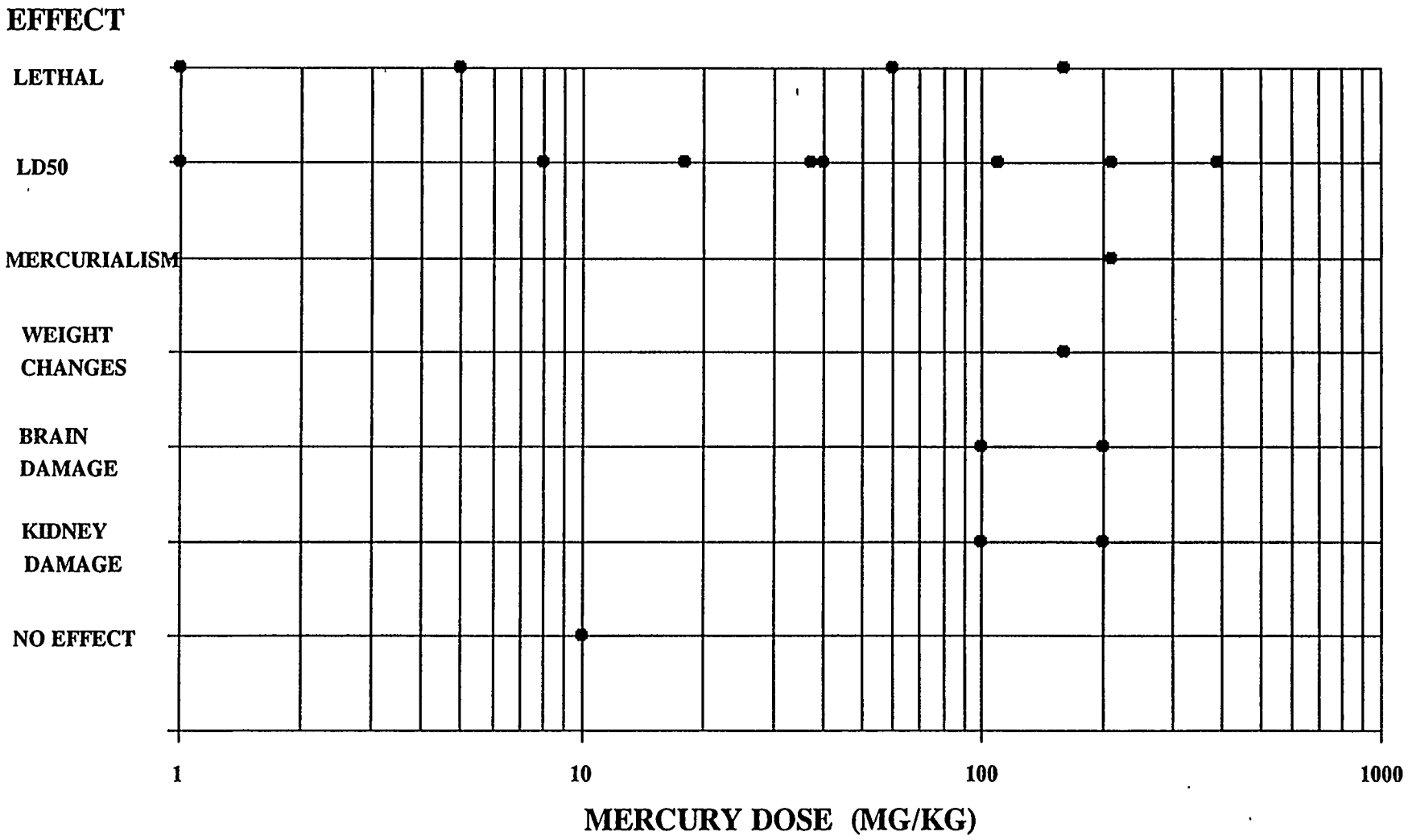

Fig. 6.56. Dose-effect pattern for mercury species and mammals.

Acute inhalation exposure to cadmium may result in pulmonary edema and death by anoxia. Other acute effects include cellular proliferation in the alveoli and hyperplasia, occasional intra-alveolar hemorrhage, peribronchial fibrosis, and emphysema. Chronic inhalation exposure to cadmium may result in proteinuria and emphysema.

Cadmium is a known animal teratogen and reproductive toxicant. It has been shown to cause renal dysfunction in both animals and humans. The progressive accumulation of cadmium has been shown to occur over the life span in animals, with the amount of accumulation related to the presence and amount of metallothionein, a cadmium binding protein. Aquatic organisms are thought to be the biotic group most sensitive to the adverse effects of cadmium. For freshwater biota in general, decreases in standing crop and growth, reproductive inhibition, immobilization, and changes in population have been associated with concentrations of 0.7 to 5 μg/L. Sublethal effects in birds have included growth retardation, anemia, and testicular damage. Mallard ducklings have exhibited altered blood chemistry and kidney lesions upon exposure to 20 mg/kg diet. Cadmium has produced teratogenic effects in amphibians, birds, and mammals. The deleterious effects of cadmium are more pronounced in the presence of mercury or lead.

Reports of questionable adequacy exist of increased incidence of tumors in animals following an oral exposure to cadmium. There is limited evidence of carcinogenicity in rats and mice by two routes.

Polychlorinated Biphenyls

PCBs are a class of chemicals of environmental and human health concern. PCBs are extremely stable compounds and are slow to chemically degrade in the environment. PCBs tend to bond tightly to particulate matter, particularly in soils and sediments of lakes, estuaries, and rivers, where they may remain available for resuspension for at least 8 to 15 years. In the water column, the hydrophobic PCBs partition into the more apolar compartments of the ecosystem or are physically adsorbed on particulate matter. High partition coefficients correlate well with PCB biomagnification in fatty tissues of aquatic organisms and with incorporation into sediments. PCB levels are usually highest in aquatic sediments containing microparticulates and high organic or clay content.

The transfer of PCBs on microparticulate materials and into phytoplankton is well documented, as is partitioning from aqueous solution into algal lipids. In phytoplankton, PCBs have been shown to exert inhibitory effects on photosynthesis and cell motility. In addition to

direct toxic effects on algae, accumulated PCBs can be introduced into the aquatic food chain through this route. In the terrestrial system, however, it is the lower chlorinated isomers (which are more water soluble and more volatile) that have been accumulated preferentially in crop plants relative to higher chlorinated isomers.

PCBs are relatively insoluble in water but are freely soluble in nonpolar organic solvents and in biological lipids. Many fish and wildlife species, including salmon, trout, turtles, eagles, herons, fish-eating birds, mink, river otters, and bats have been found to contain measurable, and in some cases potentially harmful, PCB residues, especially in adipose (fatty) tissues. Biological responses to individual isomers or mixtures can vary widely, even among closely related taxonomic species, and are also influenced by the presence of impurities in the PCB formulation, such as polychlorinated dibenzofurans (PCDFs).

Aquatic invertebrates play an important role in the cycling of PCBs within and between ecosystems. Sediment contamination has been implicated as a major source of PCB contamination. Great Lakes salmon sampled near stations with high flushing and sedimentation rates contained the lowest PCB tissue concentrations. Although diet is a major route of PCB uptake in many species of fish, there are notable exceptions, and considerable interspecies differences exist in the responses of teleosts to PCB loadings. LC^{50} concentrations for sensitive freshwater and marine organisms have been shown to vary from 0.1 to 10.0 $\mu g/L$ during exposures of 7 to 38 d. In general, toxicity increases with increasing exposure, and crustaceans and younger developmental stages are the most sensitive biotic groups.

Snapping turtles (*Chelydra serpentia*) are capable of storing high concentrations of PCBs in fat without any apparent detrimental effects and may be useful as biological indicators for lipophilic substances, including PCBs.

Residues of PCBs in birds are modified by numerous biotic factors, including fat content, tissue specificity, sex, and developmental stage. Comparative studies have found the highest PCB residues in birds with low fat content and in poor condition on capture.

In mammals, PCBs are readily absorbed through the gut, respiratory system, and skin. Initially, PCBs concentrate in liver, blood, and muscle. Eventually, accumulations are highest in adipose tissue and skin. Among mammals, the mink is especially sensitive to PCBs. Diets containing 6.7 to 8.6 mg Aroclor PCBs/kg fresh weight killed 50% of the mink in 9 months. Orally administered Aroclor PCBs resulted in LD^{50} values of 750 to 4,000 mg/kg body weight.

Signs of PCB poisoning in mink include anorexia, weight loss, lethargy, and disheveled appearance. Prior to death, dark fecal stools indicative of the presence of blood from the upper gastrointestinal tract (confirmed by necropsy) and enlarged livers have been observed (Eisler 1986).

Chlordane

Technical chlordane (CAS No. 57-74-9) consists of about 45 components, primarily *cis*-chlordane (19%), *trans*-chlordane (24%), heptachlor (10%), *cis*- and *trans*-nonachlor (7%), and various chlordane isomers (22%). The half-life of chlordane in water is comparatively short; *cis*-chlordane, for example, usually persists < 18 h in solution. In soils, however, some chlordane isomers persist for 3 to 14 years because of low solubility in water, high solubility in lipids, and relatively low vapor pressure. There seems to be little accumulation of chlordane in crops grown in contaminated soils.

Chlordane is readily absorbed by warm-blooded animals through skin, diet, and inhalation, and it is distributed throughout the body. Food chain biomagnification is usually low, except in some marine mammals.

Chlordane has been applied extensively to control pestiferous soil invertebrates, usually at rates between 0.6 and 2.24 kg/ha. Chlordane at 1.12 to 2.24 kg/ha has been shown to be lethal to fly and beetle larvae and also caused reductions in populations of various species of soil invertebrates. Many species of aquatic organisms are adversely affected at concentrations in water between 0.2 and 3.0 μg technical chlordane/L.

Sensitive bird species had reduced survival on diets containing 1.5 mg chlordane/kg in their diet, or after a single oral dose as low as 14.1 mg chlordane/kg body weight. Among nontarget soil species, earthworms are especially sensitive.

Polycyclic Aromatic Hydrocarbons

PAHs are a class of compounds consisting of substituted and unsubstituted polycyclic rings and are generated by the incomplete combustion of organic materials. Their chemical, physical, and biological properties vary with their molecular structure, geometry, and conformation. The unsubstituted lower molecular weight PAHs exhibit significant acute toxic effects to some organisms but have not been demonstrated to be carcinogenic. The higher molecular weight PAHs, containing 4 to 7 rings, have been shown to be carcinogenic, mutagenic, or teratogenic to a wide variety of organisms, including fish and other aquatic life, amphibians, birds, and

mammals. In general, PAHs do not significantly bioconcentrate up food chains despite their high lipid solubility, possibly as a result of the ability of many organisms to metabolize these substances. Inter- and intraspecies response to PAH exposure is variable, particularly under conditions of exposure to multiple PAH compounds.

PAHs are rather persistent in the environment. Some PAHs are carcinogenic, causing both systemic tumors and tumors at the site of application. Several PAHs are shown to promote carcinogenesis in experimental animals. Adverse effects on the liver and kidney are often associated with exposure to PAHs.

Very little information on specific PAHs is available. The environmental fate and transport of these compounds are largely inferred from data on benzo[a]pyrene and mixtures of PAHs. The relatively high log K_{ow} of PAHs indicates that they should be strongly adsorbed to carbonaceous sediments and soils and, hence, have a low mobility in the environment. The available information suggests that these compounds can accumulate in the sediment and biota of the aquatic environment, and that absorption is the dominant aquatic transport/fate process. Atmospheric transport of PAHs generally occurs by adsorption onto airborne particulate matter. Some volatile and relatively low molecular weight PAHs may be transported by volatilization.

PAHs are relatively insoluble in water, but the dissolved portion may undergo rapid, direct photolysis. Singlet oxygen is the oxidant, and quinones are the products of these reactions. In the presence of excessive amounts, oxidation by chlorine and ozone may also be a relevant fate process.

Although PAHs are rapidly bioaccumulated, their metabolism and elimination is fairly rapid in most organisms. Bioaccumulation, especially in vertebrates, is usually short-term, and therefore is of less importance. However, the available data indicate that higher molecular weight PAHs are metabolized slowly by microbes and more readily by higher animals. Biodegradation probably occurs more slowly in aquatic systems than in soil, and it may be much more important in systems that are chronically affected by PAH contamination.

Data on the health effects of PAHs predominantly pertain to possible carcinogenic effects. However, the application of carcinogenic PAHs on the skin in experimental animals is reported to cause destruction of sebaceous glands (skin), hyperkeratosis, hyperplasia, and ulceration. Many carcinogenic PAHs are immuno-suppressive agents. Toxic manifestations in the liver and kidney are reported in experimental animals exposed to PAHs. Workers exposed to PAH-containing materials have exhibited chronic dermatitis, hyperkeratosis, and other skin manifestations.

The potential for PAHs to induce malignant transformation dominates the consideration of health hazards resulting from exposure because there often are no overt signs of toxicity until the dose is high enough to produce a high incidence of tumor.

PAHs administered by various routes have been found to produce varying degrees of carcinogenic response depending on the type of PAH administered and the recipient animal species. The oral, topical, intraperitoneal, intratracheal, and respiratory are the frequently adopted routes for PAH administration. Skin painting (dermal exposure) experiments have revealed potent carcinogenic effects of several PAHs in the mouse. Similarly, high liver and mammary gland cancer incidence has been observed through the oral route of administration. There are several reports on the carcinogenic effects in the lungs of animals exposed to PAHs.

6.3.1.2 Toxicity-body burden data

There are a few reported instances in which a whole body burden measurement can be linked to a specific toxic effect. Although there are many instances in which mercury and PCBs have been measured in tissues of organisms in the field and in the laboratory, most of the reports give a concentration with no indication of a toxic effect. Further, some studies in which the link is shown between a concentration of a toxic substance and an effect focus on a particular organ (e.g., liver or kidney) where the toxicant accumulates.

Mercury

Data on the relationship between whole body burden of mercury and toxic effects in fish indicate these effects are species-specific. For example, rainbow trout (*Salmo gairdneri*), exhibiting loss of equilibrium and hyperplasia, had a measured body burden of 20-30 ppm (Niimi and Lowe-Jinde 1984). Conversely, brook trout (*Salvelinus fontinalis*) died with a much lower whole body burden of 5 - 7 ppm (Armstrong 1979).

There are examples of terrestrial organisms similar to those in EFPC in which both body burden and toxic effects have been simultaneously measured. Mallard ducks (*Anas platyrhynchos*) exhibited physiological and behavioral differences that could effect reproductive success at 1.4-1.5 ppm mercury in body tissues (Heinz 1979, Heinz 1980). Female ducks laid fewer eggs and laid a greater percentage of them outside their nest boxes than did control animals, resulting in fewer ducklings. Ducklings from parents fed mercury were less responsive to maternal calls and were more easily frightened than those from control parents (Heinz 1979).

PCBs

In fish, the primary biochemical effect of PCBs is the induction of hepatic mixed function oxidase (MFO) systems, which increase the organism's capacity to biotransform or to detoxify xenobiotic chemicals and endogenous steroids. Klauda et al. (1981) reported on the effects of PCBs on the liver of Atlantic tomcod, *Microgadus tomcod*. At 0.07 ppm total body burden (wet weight), they reported a normal liver. At 0.08 ppm, livers in exposed fish were hemorrhagic. In *M. tomcod* with 0.26 ppm total body burden, livers had small pustules and at 0.27, tumors were present.

In a field study, Batty et al. (1990) reported on white-footed mice (*Peromyscus leucopus*) living in an area contaminated with PCBs. Those mice with 0.24 - 4.17 ppm PCBs total body burden had liver changes and reduced reproductive success when compared to controls from uncontaminated areas.

An experimental study of mercury poisoning in manure worms (*Eisenia foetida*) showed no visible or behavioral effects at 13 and 27 ppm whole body burden (wet weight) (Beyer et al. 1985). At 85 ppm mercury whole body burden, *Eisenia foetida* exhibited reduction in segment regeneration (Beyer et al. 1985). *Eisenia foetida* exposed to PCBs exhibited immunosuppression at 1900 ppm (dry weight) and death at 3900 ppm (Fitzpatrick et al. 1992).

6.3.2 Media Toxicity Data

Toxicity tests on a variety of aquatic and terrestrial organisms using EFPC surface waters, soils, or sediments have shown deleterious effects, but have failed to provide conclusive evidence of consistent spatial patterns. Significant reductions in growth of fathead minnows were observed at sites near the Y-12 Plant, and fingernail clams showed reduced survival and reproduction at the EFPC sites nearest the Y-12 Plant and in EFPC compared with reference sites.

6.3.2.1 Historical surface water investigations

Several toxicity investigations have been conducted using EFPC surface waters (Table 6.43). The studies were conducted from March 1986 through 1990 using a variety of test organisms, including green algae, daphnia, caddisfly larvae, mollusks, and fish. Various test endpoints were used in these tests, including mortality, growth, and fecundity. Most of the studies used surface water grab samples and were conducted in the laboratory. Some of the studies were conducted in situ, and one was performed using artificial stream channels set up at various sites

Table 6.43. Summary table of media toxicity investigations conducted in EFPC

Media	Test type	Organism	Test endpoint(s)	Test dates
Surface water (grab samples)[a]	Laboratory 7-d chronic (static with renewal)	*Ceriodaphnia dubia/affinis*	Survival, fecundity	March–July 1986
Surface water (grab samples)[a]	Laboratory 7-d chronic (static with renewal)	*Pimephales promelas*	Survival, growth	March–July 1986
Surface water (grab samples)[b]	Laboratory 7-d chronic (static with renewal)	*C. dubia*	Survival, fecundity	Sept. 1986–Oct. 1988
Surface water (grab samples)[b]	Laboratory 7-d chronic (static with renewal)	*P. promelas*	Survival, growth	Sept. 1986–Oct. 1988
Surface water (grab samples)[b]	Laboratory 2-hour incubation (static)	*Haematococcus sp.*	Amount ^{14}C fixation (photosynthesis rate)	June–July 1987
Instream Surface water[b]	In situ 84-d exposure	*Sphaerium sp.*	Survival, growth	July–Oct. 1988
Instream Surface water[b]	In situ 88-d exposure	*Sphaerium sp.*	Survival, growth	Oct. 1988–Jan. 1989
Instream Surface water[c]	In situ 121-d exposure	*S. fabale*	Survival, growth, natality	1990
Surface water and stream substrate[b]	On-site, artificial channel, flow-through	*Hydropsyche depravata*	Survival, growth	Sept.–Dec. 1988
Surface water and stream substrate (grab samples)[b]	Laboratory exposure, constant temperature	*H. depravata*	Growth emergence	Sept.–Dec. 1988

[a]Loar 1992
[b]Hinzman 1992
[c]Kornegay 1991

along EFPC. Effects to biota generally were more severe for individuals in EFPC closer to the Y-12 Plant, with fingernail clams exhibiting the highest sensitivity to water-borne impacts.

Two concurrent series of 7-d toxicity tests were conducted during March and June through July 1986 as part of the Y-12 BMAP (Loar 1992) to estimate chronic toxicity. These tests used surface water from ten sites in EFPC below New Hope Pond. Mercury or PCB concentrations were not measured in the water samples. The ten sites spanned most of the length of EFPC before its confluence with Poplar Creek, and included EFK 23.7, EFK 22.8, EFK 21.9, EFK 20.5, EFK 18.2, EFK 16.1, EFK 13.8, EFK 10.9, EFK 7.6, EFK 5.1, and EFK 2.1. Sites at EFK 18.2, EFK 13.8, and EFK 10.9 corresponded to existing BMAP sampling locations for various aquatic biota. *Ceriodaphnia* (*Ceriodaphnia dubia/affinis*) and fathead minnows (*Pimephales promelas*) were the test organisms. Test endpoints were survival and fecundity for the *Ceriodaphnia* and survival and growth for the fathead minnow. No statistically significant decreases in survival or fecundity of *Ceriodaphnia*, or survival or growth in fathead minnows, was observed at the site closest to the Y-12 Plant (EFK 23.7) during the first series of tests, but a 10% reduction in survival of *Ceriodaphnia* was observed for sites EFK 16.1, ERK 13.8, and EFK 5.1 (Table 6.44). During the second series of tests, survival of *Ceriodaphnia* decreased 10% at EFK 18.2, EFK 13.8, and the control site. A downstream reduction in *Ceriodaphnia* fecundity was not observed during either set of tests. Statistically significant reductions in growth of fathead minnows were observed for sites EFK 22.8 (SAIC Site 1) and EFK 18.2 [$P < 0.05$, analysis of variance (ANOVA) Dunnett's one-tailed test] during the first set of tests, and for sites EFK 22.8 (SAIC Site 1), EFK 21.9 (SAIC Site 2), EFK 20.5, and EFK 7.6 during the second set of tests. No reduction in survival of fathead minnow larvae was observed during either set of tests.

During October 1986 through October 1988, eight additional series of water quality and 7-d chronic toxicity tests were conducted using *Ceriodaphnia* and fathead minnows, and surface water grab samples were taken from six sites in EFPC: EFK 22.8, EFK 21.9, EFK 20.5, EFK 18.2, EFK 13.8, and EFK 10.9 (Hinzman 1992). Test endpoints were survival and fecundity for *Ceriodaphnia* and survival and growth for the fathead minnow. Mercury and PCBs were not measured in the water samples, but total residual chlorine concentration was measured daily during the toxicity tests (Table 6.45). Mean concentrations of total residual chlorine did not exceed the U.S. EPA criteria for protection of aquatic life (16.5 μg/L) [11 μg/L, 4-d average (EPA 1986b)] at any of the sites and were less than the concentration reported by Arthur and Eaton (1971) to have no significant impact on egg production in fathead minnows. In general, the tests provided little evidence for systematic changes in water quality with downstream distance

Table 6.44. Toxicity test results for water samples collected daily from ten sites on EFPC in 1986

SITE	March 6 - March 13				June 26 - July 3			
	Ceriodaphnia		Fathead Minnow		*Ceriodaphnia*		Fathead Minnow	
	Surv.[a]	Repro[b]	Surv.	Growth	Surv.[a]	Repro[b]	Surv.	Growth[c]
EFK 23.7	100	27.6	90.0	0.564	90	27.8	97.5	0.432
EFK 22.8	100	28.9	92.5	0.382[d]	100	24.8	92.5	0.347[d]
EFK 21.9	100	30.3	95.0	0.515	100	21.5	92.5	0.355[d]
EFK 20.5	100	30.6	92.5	0.536	100	22.8	92.7	0.197[d]
EFK 18.2	100	29.8	92.5	0.425[d]	90	21.4	100.0	0.433
EFK 16.1	90	37.0	87.5	0.481	100	25.1	95.0	0.440
EFK 13.8	90	34.5	90.0	0.583	90	24.5	90.0	0.426
EFK 10.9	100	23.3	90.0	0.468	100	16.4	97.5	0.517
EFK 7.6	100	30.2	80.0	0.495	100	25.1	97.5	0.266[d]
EFK 5.1	90	28.6	85.0	0.479	100	19.2	95.0	0.533
EFK 2.1	100	28.6	87.5	0.639	100	34.0	90.0	0.384
Control	100	28.6	100.0	0.571	90	14.2	95.0	0.553

[a] Percentage survival is based on ten replicates (one animal per replicate) for *Ceriodaphnia*, and on four replicates (ten animals per replicate) for fathead minnow larvae.

[b] Reproduction of *Ceriodaphnia* is the mean number of offspring per female for the 7-d test, with reproduction of females that die before leaving offspring being set to zero.

[c] Growth of fathead larvae (average mg per animal over the 7-d test) is corrected for the initial average weight of the larvae based on a representative subsamples of larvae at the start of the test.

[d] Test endpoints that differed significantly from control [$p < 0.05$; SAS-GLM (ANOVA) followed by Dunnett's one-tailed test].

Source: with permission Loar (1992)

Table 6.45. Summary statistics (mean, SD, range, and number of observations) for pH, alkalinity (mg CaCO/L), free chlorine (μg/L), and total residual chlorine (μg/L) for water samples collected from six sites in EFPC during the eight 7-d toxicity tests conducted between October 7, 1986 through October 6, 1988.

Parameter	EFPC Site (km)					
	22.8	21.9	20.5	18.2	13.8	10.9
pH	8.07 ± 0.10 7.84 - 8.26 (56)	8.05 ± 0.10 7.89 - 8.30 (56)	8.09 ± 0.10 7.88 - 8.30 (56)	8.08 ± 0.10 7.85 - 8.30 (55)	8.10 ± 0.09 7.79 - 8.31 (56)	7.98 ± 0.10 7.79 - 8.19 (56)
Alkalinity	112.6 ± 8.2 86 - 128 (56)	113.4 ± 9.1 79 - 127 (56)	114.9 ± 8.0 82 - 129 (56)	119.3 ± 9.2 78 - 138 (55)	120.9 ± 8.9 86 - 132 (56)	128.9 ± 12.8 84 - 192 (56)
Free chlorine	3 ± 9 0 - 30 (10)	0 - (2)	0 - (2)	- - -	0 - (1)	4 ± 10 0 - 30 (17)
Total Residual Chlorine	3 ± 12 0 - 50 (50)	1 ± 3 0 - 20 (50)	<1 ± 1 0 - 10 (50)	0 - (49)	<1 ± 3 0 - 20 (50)	11 ± 17 0 - 50 (50)

Source: Adapted with permission Hinzman (1992).

from New Hope Pond. Longitudinal differences in water quality resulting in growth or fecundity changes smaller than about 20 to 30% of the maximum value for the parameter at any site could not be detected statistically (Hinzman 1992). To determine if there were site-to-site concordance of response patterns for the two species, response ranges of fish growth and *Ceriodaphnia* fecundity were expressed as percentages of the highest value among all the sites for that particular test. The sites were ranked with respect to the number of times they were the "best" (highest percentage) or "worst" (lowest percentage) for each response variable. Rankings showed no detectable longitudinal pattern in EFPC for either fathead minnow growth or *Ceriodaphnia* fecundity in the 7-d tests (Hinzman 1992). However, EFK 13.8 appeared to be consistently better than the other sites because it never ranked lowest for either species and ranked highest for at least one species in six of the eight tests.

Photosynthesis rate of the green algae *(Haematococcus)* was measured by ^{14}C uptake rate of cultures inoculated in water samples collected from nine sites in EFPC (EFK 22.8, EFK 21.9, EFK 20.5, EFK 18.2, EFK 16.1, EFK 13.8, EFK 10.9, EFK 7.6, and EFK 5.1) and one reference site in Brushy Fork (Hinzman 1992). The tests were conducted on six dates during June and July 1987. Although rates of ^{14}C incorporation were significantly different among the sites ($P < 0.05$, Duncan's Multiple Range Test), no clear longitudinal pattern was observed. Highest ^{14}C incorporation rates were obtained in water from EFK 10.9, whereas lowest rates were at EFK 20.5, EFK 18.2, and EFK 13.8. Interestingly, water from the sites closest to the Y-12 Plant (EFK 22.8 and EFK 21.9) yielded intermediate rates of ^{14}C incorporation in *Haematococcus*. Hinzman (1992) was unable to identify any site-specific water chemical factors that could account for the differences in the rate of ^{14}C incorporation in *Haematococcus*.

During September through December 1988, the Y-12 Plant BMAP conducted two media toxicity tests using caddisfly larvae *(Hydropsyche depravata)* (Hinzman 1992). The first set of tests were inconclusive because the channels at EFK 23.1 received so much silt during storm overflows that most of the experimental organisms were lost after six weeks. The second study using *H. depravata* consisted of a constant-temperature laboratory exposure using ambient water from the same two sites in EFPC, using dechlorinated tap water as a control. Growth and presence or absence of emergence were the test endpoints. No significant differences in growth rates or emergence were observed among groups of individuals maintained in water from EFK 23.1, from EFK 14.0, or the tap water control (P level not stated; one-way ANOVA).

Toxicity studies using fingernail clams (*Sphaerium fabale*) were conducted by the Y-12 Plant BMAP during July 1988 through January 1989 (Hinzman 1992). The first set of toxicity tests was conducted at two sites in EFPC (EFK 23.4 and EFK 13.8) and at the reference site,

Brushy Fork, using two size classes of clams in plastic cages during an 81-d exposure. The smallest size class consisted of clams with shell lengths ranging from 7.5 to 8.5 mm (~0.3 in.) (measured as greatest anterior-to-posterior length), and the large size class had shell lengths ranging from 10.7 to 11.9 mm (0.4 to 0.5 in.). After 81 d, survival of the small and large size clams at Brushy Fork was at 87% and 100%, respectively. However, survival of the large clams after 81 d at EFK 13.8 and EFK 23.4 was only at 41% and 8%, respectively. At EFK 23.4, no small clams were alive after 81 d. At EFK 13.8, survival of small clams was at 100% after 38 d, but the tray holding the clams was lost after the 38th day during high flows. Growth rates of clams from both size classes were greatest at Brushy Fork. After 81 d, little or no growth was observed in large clams at either EFK 23.4 or EFK 13.8, or in small clams at EFK 23.4. No explanations were provided as to the cause(s) of the low survival or growth rates, but environmental conditions at EFK 23.4 (near the New Hope Pond outfall) were clearly more deleterious to the clams than were conditions either at EFK 13.8 or at the Brushy Fork reference site.

The second set of clam toxicity tests was conducted during October through December 1988, at the same two sites in EFPC and the Brushy Fork reference site, and two additional reference sites, Hinds Creek at kilometer 20.6, located in northeast Anderson County, Tennessee, and Bull Run Creek at kilometer 20.0, located in Union County, Tennessee. Only one size class of clams (7.25 to 8.3 mm (~0.3 in.) in shell length) was used. Survival of clams at EFK 23.4 was at only 4% after 88 d, but no explanations were given for the high mortality. At EFK 13.8, survival of the clams was at 94% after 88 d, which was similar to the 100% survival rate at all three reference sites.

During 1990, the Y-12 Plant BMAP conducted studies to evaluate natality of fingernail clams as an additional indicator of water quality at the two sites in EFPC (at EFK 23.4 and 13.8), as well as at reference sites in Brushy Fork and Beaver Creek (Kornegay 1991). Each clam was placed in a small plexiglass cylinder covered with mesh at both ends. At approximately 2-week intervals during a 121-d exposure, the number of offspring were counted in each cylinder, and mortality of adults was noted. In addition, lengths of the adults were measured. Survival of clams at the reference sites was at 100%, but only 65.2% and 82.6% at EFK 23.4 and EFK 13.8, respectively. Growth rates of clams at Beaver Creek and EFK 23.4 were approximately three-fold greater than at EFK 13.8. The highest total and mean number of offspring were observed at the Brushy Fork reference site (80 and 3.8, respectively), followed by the Beaver Creek reference site (44 and 2.1), EFK 13.8 (35 and 1.6), and EFK 23.4 (22 and 1.2). Therefore,

reproduction of clams at the two sites in EFPC was lower than at the two reference sites and was lowest at the site nearest the New Hope Pond outfall.

6.3.2.2 Historical soil/sediment ingestion investigations

A soil/sediment toxicity study was conducted by Revis et al. (1989b), using mice to determine potential health risks from eating mercury-contaminated soils and sediments collected from eight locations in EFPC. Total mercury concentrations in the soils and sediments ranged from 2 to 1800 mg/kg (using nitric acid extraction). Freeze-dried soil samples were incorporated into mouse feed pellets (5% by weight) and fed to Swiss mice over a 20-month period. Some of these soil samples were also spiked with mercuric chloride before being incorporated into the mouse feed pellets, resulting in final concentrations of mercury ranging from 500 to 8000 mg/kg. After 6, 12, and 20 months of exposure, a sample of animals from each group was weighed, given a swim test to evaluate neurological performance, and then sacrificed. Livers and kidneys were examined for histopathological damage and analyzed for mercury. At 12 months, urine was collected for 24 h from survivors and analyzed for protein, creatinine, and gamma-glutamyl transferase (gamma-GT), indicators of kidney function. In addition, a short-term feeding study was carried out to determine the initial rate of mercury absorption from soil-contaminated feed. Previously unexposed mice were fed diets of contaminated soil for 24 h, feces were collected for 96 h and then analyzed for mercury, and the amount of excreted mercury was correlated with the calculated amount of ingested mercury.

Mercury concentrations in liver and kidney tissues were highly correlated with diet mercury concentrations. However, pathological morphological effects to the kidneys were not observed, except in the groups of mice fed soil spiked with mercuric chloride concentrations of 4000 and 8000 mg/kg. In addition, biochemical measures of kidney damage (proteinuria changes in creatinine or gamma-GT) were not significant and were not correlated with diet, kidney, or liver mercury concentrations. No significant correlation was observed between diet mercury concentrations and changes in body weight, mortality, or teratogenic effects. The results of the Revis et al. (1989b) study are pertinent to this ecological risk assessment because several small rodents are indicator organisms for this ERA.

6.3.2.3 Fish corral study of mercury bioavailability

The contamination of upper EFPC by historical and continuing releases of inorganic mercury from the Y-12 Plant has resulted in high mercury accumulation by fish (Kornegay 1991). A key factor in determining cost-effective corrective measures for this area of EFPC is the relative importance of continuing waterborne mercury releases versus sediment-bound mercury

in sustaining unacceptable mercury concentrations in fish. Studies by Elwood et al. (1988) suggest that ongoing waterborne releases are the most significant contribution to the bioaccumulation of mercury in biota. In support of the CERCLA remedial investigation/feasibility study for EFPC, the Environmental Restoration Division of the Oak Ridge Y-12 Plant is supporting field investigations to resolve the issue of the bioavailability of sediment-bound mercury. The first phase of these investigations was conducted in upper EFPC downstream of the Y-12 Plant during October 1991 through February 1992. This initial investigation is summarized below. These findings are discussed in more detail in a draft report included as Appendix Q.

Four 96-in. diameter pipe enclosures were set on the concrete floor of the inlet channel of Lake Reality, two receiving mercury-contaminated water (at 1 μg/L) from EFPC and two receiving local groundwater of similar chemical content but with a lower mercury concentration (0.02 μg/L). Each enclosure isolated an area of the channel and the in situ sediment. In addition, two 500-gal polytanks were set next to EFPC, one receiving creekwater from the channel and one receiving groundwater. Hatchery-raised bluegill-green hybrid sunfish of similar size were placed in the enclosures, 30 in each pipe enclosure and 15 in each polytank. Fig. 6.57 depicts the experimental procedure.

Fish from the initial stock were analyzed for total mercury concentrations, and five fish from each enclosure were sacrificed at 3-week intervals for determination of total mercury and methylmercury concentrations. Samples of the flora and fauna that constituted the food supply for the fish were analyzed for total mercury and methylmercury concentrations several times during the course of the experiment, and water samples from both inlets and outlets of the enclosures were analyzed weekly for total mercury, methylmercury, and other chemical and physical properties. Water chemistry was monitored for only 9 weeks, but fish sampling was continued in the pipe enclosures for 9 additional weeks. Sediment cores from pipe enclosures were separated into sediment and interstitial water. Air-dried and pulverized sediment and porewater were then analyzed.

Mercury uptake rates for fish, presented in Table 6.46, were calculated by linear regression of total mercury concentration in fish (μg/g wet weight) versus time (in h). The uptake rates were statistically significant for all treatments that received creekwater; however, rates for all treatments that received groundwater were not significant. Although the mercury accumulation rates in fish were low in all four treatments, accumulation rates were lowest in the groundwater treatments. Fish in the polytank exposed only to creekwater had the highest accumulation rate.

Fish Corral Study – Experimental Design

Treatment

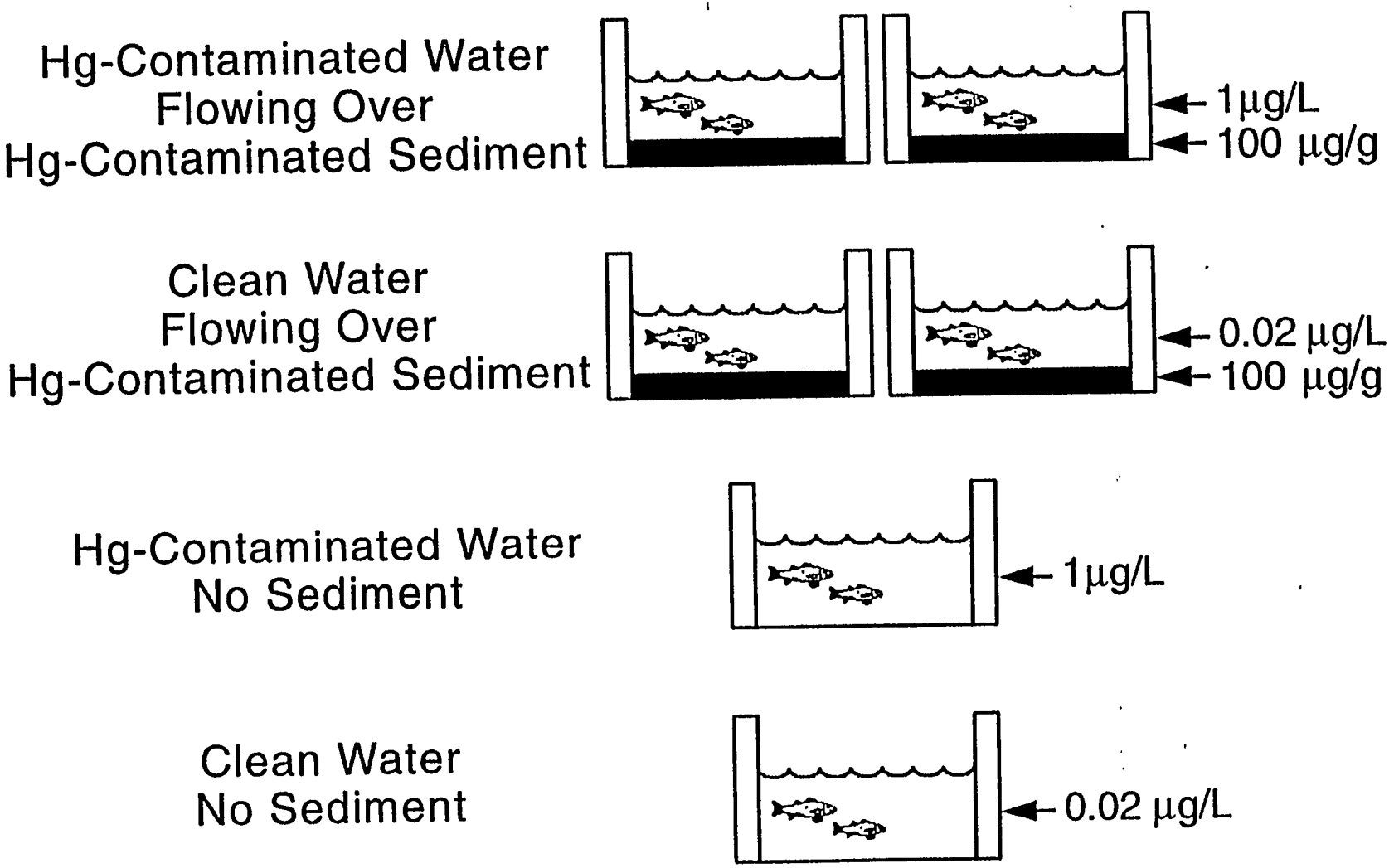

Fig. 6.57. Experimental design of fish enclosure study to determine bioavailability of mercury in Lake Reality sediments and water.

Table 6.46. Mercury uptake rates for hybrid sunfish in corrals[a]

Treatment	Exposure period		
	9 weeks	15 weeks	18 weeks
Groundwater + sediment	0.26	0.39	0.38
Groundwater + sediment	0.56	0.16	0.09
Creekwater + sediment	1.5	1.9	1.5
Creekwater + sediment	1.1	1.7	1.1
Groundwater only	0.90	—	—
Creekwater only	4.3	—	—
Creekwater + sediment (Elwood et al. 1988)		21	

[a]Mercury accumulation is measured in $\mu g\ g^{-1}\ h^{-1} \times 10^{-5}$.

In this experiment, mercury concentrations in the sediment were high, but there was no evidence that sediment-associated mercury was a direct or indirect source of mercury accumulation in the fish. Fish uptake rates were lower in the sediment-containing enclosures than in the enclosures containing water only. If ingestion of sediment with food had been a major source of mercury bioaccumulation, then uptake rates in all treatments with sediments would have been similar or higher than those treatments without sediment.

These results suggest that sediments may have been a sink rather than a source of mercury. Concentrations of dissolved mercury were lower in the creekwater-sediment treatment (Table 6.47) than in the creekwater-only treatment, suggesting that particulate materials and plants sequester dissolved mercury. The higher accumulation rate in fish in the groundwater-only treatment than in the groundwater-sediment treatment also suggests that sediment and plants are not sources of mercury for fish, but rather remove it from water.

The primary source of mercury accumulated by fish in this experiment appeared to be dissolved inorganic mercury (Table 6.47). Dissolved mercury, highest in the creekwater-only treatment where fish accumulation rates were highest, was low in groundwater treatments where accumulation rates were lower. Estimates of fish uptake rates, based on laboratory-observed uptake rate coefficients for mercury in fish and on the measured concentrations of dissolved mercury in the treatments waters, were close to the observed rates in Table 6.46, and the differences in estimated uptake rates among treatments were comparable to observed differences.

The differences in concentrations of dissolved methylmercury among treatments were not great (Table 6.47). Estimates of uptake rates for fish based on methylmercury uptake rate coefficients and measured concentrations for dissolved methylmercury were below the observed rates. Although methylmercury comprised more than 80% of the mercury in fish muscle for all other treatments, methylmercury accounted for only 28% of the total mercury in fish for the creekwater-only treatment where uptake rates for fish were highest.

Food containing methylmercury was also a likely source for mercury accumulation in fish in this experiment. Animal prey contained substantial amounts of total mercury and equivalent amounts of methylmercury, but this food source was rapidly depleted by the fish. Plant food contained very high total mercury levels but little methylmercury. Estimates of accumulation rates from mercury in plant food were very high compared with observed uptake rates; it was concluded that the assimilation efficiency of mercury from this pathway must be low. Estimates of accumulation rates from methylmercury in prey, as well as estimates of accumulation rates from methylmercury in plant food, were high enough to account for the observed rates of

Table 6.47. Mean aqueous concentrations of mercury species in fish corrals, October–December 1991[a]

Mercury species	Treatment			
	Groundwater + sediment	Creekwater + sediment	Groundwater only	Creekwater only
Total Hg	290	1200	12	1200
Dissolved Hg	23	160	6	640
Total CH_3Hg	0.2	0.46	0.034	0.15
Dissolved CH_3Hg	0.045	0.13	0.026	0.073

[a]Mercury species are measured in mg/L.

mercury accumulation in fish. However, these estimates did not explain the observed differences in mercury bioaccumulation among treatments.

Extrapolation of the observed mercury accumulation rate for fish in the creekwater-sediment treatment to a year of exposure predicts a mercury concentration in sunfish that is only 20% of the average mercury concentration of sunfish living in the upper part of the creek (measured by Kornegay 1991). Explanations for the low rate of mercury accumulation in this experiment include insufficient animal prey for food and seasonal variation in rates of methylmercury production and bioaccumulation, with greater accumulation occurring in summer.

During June through August 1992, this experiment was repeated with modifications. The initial biomass of fish per enclosure was reduced to levels close to that characteristic of ponds and reservoirs, and the size of the polytank enclosures was increased to allow more direct comparison of these water-only enclosures to water-sediment enclosures. In order to sample fish food organisms without limiting predation by fish, two additional enclosures, one for each water type, were not stocked with fish. When results of this investigation are available, they will be included in future drafts.

6.3.3 Current Biological Survey Effects Data

The results of the biological surveys whose purpose and design were discussed in Sect. 6.1 can be summarized as follows. The number of indicator fish species, benthic macroinvertebrate families, and insect adult individuals at EFPC generally increased with increasing distance downstream from the Y-12 Plant. Compared with a reference site, EFPC had fewer species of fish, families of benthic macroinvertebrates, and mean numbers of individuals per sample of adult insects with terrestrial larval forms. The number of fish species classified as tolerant of degraded conditions decreased downstream in EFPC. Taxonomic diversity (H′) of fish, benthic macroinvertebrates, and adult insects increased downstream, except at Site 3, and were lowest at the 3 sites where surface water mercury concentrations were greatest. Taxonomic diversity for fish, benthic macroinvertebrates, and adult insects with aquatic larval forms at EFPC sites were generally less than at the reference site. The richness of families from sensitive orders of Arthropoda—Ephemeroptera, Plecoptera, and Trichoptera—was lowest at the EFPC site nearest the Y-12 Plant and was lower at all EFPC sites than at the reference site. Studies of periphyton colonization and growth, populations of small mammals, earthworms, birds, and terrestrial vegetation showed less obvious patterns. For example, terrestrial arthropod diversity increased slightly as a function of increasing distance from the Y-12 Plant. A survey of EFPC in the fall identified 43 species of birds, 7 of which were classified as common year-round residents.

White-footed mice were captured at all EFPC sites except Site 6. Shrews, a vole, and a chipmunk were captured at one or more of the sites furthest from the Y-12 Plant. A limited vegetation survey and earthworm population samples revealed few patterns associated with exposure to contaminants released from the Y-12 Plant. For example, germination may be suppressed at part of Site 3.

6.3.3.1 Fish survey

Quantitative sampling of fish at six sites in lower EFPC and at a reference site in Hinds Creek was conducted during October 7 through 12, 1991, to obtain data on species richness and composition, density, diversity, and possible age structure of indicator species.

Sampling methods

All fish sampling was conducted by electrofishing according to *TP-309-5: Fish Sampling in Streams* (LWA 1990).

Sampling reaches ranged from 90 to 114 m in length (Table 6.48) and were blocked at each end with 0.64-cm-mesh seines to minimize fish escape. Sampling sites included areas subsequently sampled for benthic macroinvertebrates and periphyton and either overlapped or were within 100 m (330 ft) of the EFPC terrestrial biota sampling sites. The sampling crew consisted of two electrofishing unit operators and three or four persons with hand nets or buckets to retrieve the stunned fish. Three sampling passes were usually performed at each site, always proceeding upstream. Sampling duration was recorded after completion of each pass. Water turbidity was allowed to dissipate before subsequent passes were made in order to maintain acceptable conductivity for electrofishing and visibility. Captured fish were kept in 5-gal plastic buckets filled with water from the sample site.

Processing of the fish began before completion of each sampling pass. Each fish was identified as to species (unidentifiable individuals were wrapped in aluminum foil and kept on ice until their preservation in 80% ethanol upon return from the field), weighed on an Ohaus Lume-O-Gram™ scale to the nearest 2 g, which was accurate enough for the weight class analysis, and measured for total length. Scales were collected from redbreast sunfish (*Lepomis auritus*), stonerollers (*Campostoma anomalum*), and largemouth bass (*Micropterus salmoides*), and saved in scale envelopes for age determination analyses. Gross observations on external anomalies, such as deformities, parasites, or disease, were also recorded. All of the redbreast sunfish, stonerollers, largemouth bass, and aquatic crayfish that were captured from each of the sample reaches were saved for laboratory analysis for contaminants of concern. The organisms were

Table 6.48. Stream physical and morphological characteristics at the aquatic biota sampling sites in EFPC and Hinds Creek

Parameter	Site						
	1	2	3	4	5	6	HC3
Sample reach length (m)	98	114	114	107	99	102	90
Mean width (m)	6.2	5.6	5.9	6.6	8.1	13.7	5.7
Pool/riffle ratio	1.32	5.20	12.21	8.52	4.25	—[a]	11.84
Surrounding land type	forest[b]	forest[c]	other[d]	other[e]	forest[c]	forest[c]	forest[c]
Canopy cover	mostly covered	mostly covered	mostly covered	mostly open	mostly covered	mostly covered	mostly open
Turbidity	clear	clear	slightly turbid	clear	clear	clear	clear
Water odor	none	none	none	none	none	none	none
Water surface oils	none	none	none	none	none	none	none

[a]Not calculated - only one riffle and none downstream for >200 m.
[b]Mixed mesophytic.
[c]Bottomland hardwoods.
[d]South floodplain (to edge) is old field with scattered trees; north floodplain is field with few scattered trees and shrubs, especially at creek edge.
[e]North side is residential, south side is a narrow strip of forest and golf course.

sorted by species, wrapped in aluminum foil, and kept on ice until completion of all passes for the site. After all passes were completed, the composite samples were packed in chemically clean glass jars, sealed with custody tape, and kept in ice until stored and locked in a freezer. All remaining fish were then returned to the stream and the blocking nets were removed. At the Radian warehouse, redbreast sunfish fillets were cut from individuals that were previously wrapped in aluminum foil and covered with ice. Results of body burden analysis are provided in Sect. 6.2.

Several physical and chemical parameters were measured at each sampling site (Tables 6.48 and 6.49). Predominant surrounding land types, canopy cover, turbidity, water color and odors, and water surface oils were recorded. The length, width, and depth of the sampling reach were measured. Widths were measured at 4.88-m (16 ft) interval transects, and depths were measured at the one-quarter-mark, the midstream, and the three-quarter-mark locations on each transect.

Water samples were collected from each sample site during October 8 through 10, 1991, according to procedures detailed in *Phase Ib Sampling and Analysis Plan for Soil, Sediment, and Water* (Radian 1993a), for laboratory analysis of CLP volatile and semivolatile organics, CLP metals, isotopic uranium, gamma levels, alkalinity, sulfates, chlorides, nitrates and nitrites, dissolved orthophosphate, total organic carbon (TOC), dissolved organic carbon, and biological oxygen demand. Temperature and pH were measured with a Hannah HI 9025 pH meter. Dissolved oxygen (DO) was measured with a YSI Model 57 meter, and conductivity was measured with an Extech Oyster™ conductivity meter.

Population analysis methods

Species population estimates were made using the method of Carle and Strub (1978). Density estimates for each site were expressed as number of fish per 10 m^2. Sample area was calculated by multiplying the study reach length by the mean width.

Species diversity (H′) was calculated using the Shannon-Wiener Index:

$$H' = -\sum_{i=1}^{n} p_i \log p_i$$

where p_i equals the proportion of species i in the sample. The Shannon-Wiener Index incorporates two components of diversity: the number of species (n) and the evenness of their abundances.

Table 6.49. Water quality summary for fish sampling sites in EFPC and Hinds Creek (Oct. 8-10, 1991)

Parameter	Site						
	1	2	3	4	5	6	HC3
Alkalinity (mg/l as $CaCO_3$)	135	129	141	154	130	137	201
Biological oxygen demand (BOD) (mg/l)	<5.0	—[a]	<5.0	—[a]	<5.0	5.7	5.0
Conductivity (μmhos/cm)	470	480	480	480	430	400	380
Dissolved organic carbon (DOC) (mg/l)	1.2	1.0	1.4	3.0	2.5	2.3	1.9
Dissolved Oxygen (mg/l)	7.5	6.2	5.6	8.4	11.4	11.2	11.0
pH	8.17		7.65	7.60	7.52	7.56	8.16
Temperature (°C)	19.7	17.9	15.2	17.2	15.9	14.0	9.9
TOC (mg/l)	22.0	3.9	13.0	13.0	11.0	2.9	8.0
Total suspended solids (TSS) (mg/l)	<4.0	<4.0	<4.0	<4.0	<4.0	9.6	10.8
Calcium (mg/l)	56.2	56.8	55.7	54.0	46.5	46.4	45.3
Magnesium (mg/l)	11.0	11.6	11.5	11.2	10.6	10.3	18.6
Potassium (mg/l)	1.8	2.2	2.3	5.4	4.4	4.5	2.5
Sodium (mg/l)	14.0	14.1	12.6	21.7	18.1	17.5	2.3
Chloride (mg/l)	15.6	15.7	16.4	20.7	17.1	16.2	2.7
Nitrate/nitrite (mg/l)	3.3	3.3	3.1	6.0	0.26	0.41	0.50
Orthophosphate (mg/l)	0.24	0.26	0.24	1.46	1.25	1.20	0.01
Sulfate (mg/l)	67.4	71.6	64.7	55.5	44.2	38.3	11.8
Lead (μg/l)	3.10	<2.00	3.10	<2.00	2.90	2.40	<2.00
Mercury (μg/l)	0.54	0.43	0.32	<0.20	<0.20	<0.20	<0.20

[a]Invalid analysis

Results

Environmental conditions. Stream physical and morphological characteristics for the EFPC and Hinds Creek sites are summarized in Table 6.48. Surrounding vegetation at most sites was bottomland hardwood forest. However, Site 3 was bordered by old fields and scattered trees and shrubs, and Site 4 was bordered by residential areas to the north and by a narrow strip of bottomland hardwood forest along a golf course to the south. No unusual water odors or surface water oils were observed at any of the sites. The canopy was mostly closed at all sites except Site 4 and Hinds Creek, where it was mostly open. Mean width among all sites except sites 5 and 6 was similar, ranging from 5.6 to 6.6 m (18.5 to 21.8 ft). Mean widths at Sites 5 and 6 were 8.1 and 13.7 m (26.7 and 45.2 ft), respectively.

Water quality characteristics at the sampling sites are summarized in Table 6.50. The dissolved oxygen concentration at Site 3 was only at about 55% saturation, noticeably lower than at all other sites. The reduced dissolved oxygen concentration would not threaten resident fish populations, but could result in increased respiratory stress and potential synergistic effects with toxicants. The highest total mercury concentration in surface water was 0.54 μg/L, measured at Site 1. Total surface water mercury concentrations were 0.43 μg/L at Site 2, 0.32 μg/L at Site 3, and <0.20 μg/L at the remaining EFPC sites and at Hinds Creek.

Species richness, composition, and relative abundance. Twenty-two species plus one hybrid sunfish were collected from the six sites in EFPC, compared to fourteen species from the single sampling site at Hinds Creek (Table 6.50). Table 6.51 summarizes the family names, common and scientific names, relative abundances, population estimates and 95% upper-bound confidence limits, tolerance and trophic classifications, and lithophilic spawner information (i.e., regarding species that release their eggs in gravel, provide no parental care, and are affected by increases in siltation and pollutants) for all fish species captured in EFPC and Hinds Creek. Only one of the species collected at the reference site [the stripetail darter (*Etheostoma kennicotti*)] was not captured at any of the EFPC sites. However, ten species were captured in EFPC that were not found at the reference site (Table 6.52). Seven of the 10 species were classified as tolerant of degraded water quality conditions, in contrast to 3 species that were classified as moderately intolerant. The number of species captured at each of the 6 EFPC sample sites ranged from 8 to 12 and was always less than at the reference site. Sites 2 and 3 yielded the fewest species (43% and 37% less than the reference site, respectively), whereas the remaining EFPC sites each yielded 22% or fewer species than the reference site. The number of species captured increased progressively downstream, from 8 at Site 2 to 12 at Sites 5 and 6.

Table 6.50. Fish sampling summary for sites in EFPC and Hinds Creek (Oct.7-12, 1991)

Parameter	Site						
	1	2	3	4	5	6	HC3
Sampling area (m^2)	610	655	670	699	815	1403	517
Sampling duration (min)	126	114	102	150	102	1197	127[a]
Total number of species	12	8	9	11	12	12	14
Total number of captured individuals	690	238	242	165	78	59	366
Estimated population size[b]	998	285	1108	194	109	60	483
Estimated population density (individuals/10 m^2)	16.35	4.35	16.55	2.78	1.34	0.43	9.34
H'[c]	0.530	0.537	0.506	0.736	0.778	0.848	0.771
% Tolerants[d]	73	63	56	36	25	17	29

[a]Estimated; pass one was 67 min.; pass two estimated 60 min.
[b]Methods of Carle and Strub (1978).
[c]H' = fish species diversity (Shannon-Weiner Index).
[d]Percentage of captured species classified as tolerant of degraded water quality conditions (Ohio EPA 1989).

Table 6.51. Summary Table listing fish family, scientific and common name, relative abundance, tolerance classification, trophic feeding group, and lithophilic spawner information for fish collected for EFPC and Hinds Creek during October 7-12, 1991

Species		Site 1	2	3	4	5	6	HC3	Tolerance Classification	Trophic Group	Lithophilic Spawner?
Clupeidae	Rel. Abund.	0.00	0.00	0.00	0.00	2.60	0.00	0.00			
Gizzard shad	Population	b	b	b	b	2.00	b	b	-	GEN	NO
(Dorosoma cepedianum)	95% Upper	b	b	b	b	a	b	b			
Cyprinidae	Rel. Abund.	0.00	0.00	0.00	0.00	0.00	20.30	0.80			
Bluntnose minnow	Population	b	b	b	b	b	12.00	4.00	TOL	OMN	NO
(Pimephales notatus)	95% Upper	b	b	b	b	b	12.00	a			
Blacknose dace	Rel. Abund.	3.80	6.30	1.20	1.20	0.00	0.00	3.00	TOL	GEN	YES
(Rhinichthys atratulus)	Population	38.00	17.00	3.00	2.00	b	b	16.00			
	95% Upper	86.00	36.32	a	3.04	b	b	a			
Creek chub	Rel. Abund.	0.10	3.80	2.10	1.20	0.00	0.00	2.20	TOL	GEN	NO
(Semotilus atromaculatus)	Population	1.00	9.00	6.00	2.00	b	b	8.00			
	95% Upper	1.27	9.99	a	2.00	b	b	9.61			
Fathead minnow	Rel. Abund.	1.00	0.00	0.00	0.00	0.00	0.00	0.00	TOL	OMN	NO
(Pimephales promelas)	Population	7.00	b	b	b	b	b	b			
	95% Upper	16.59	b	b	b	b	b	b			
Rosefin shiner	Rel. Abund.	0.00	0.00	0.00	0.00	0.00	11.90	0.20	MIN	INS	YES
(Notropis ardens)	Population	b	b	b	b	b	7.00	1.00			
	95% Upper	b	b	b	b	b	7.00	2.10			
Striped shiner	Rel. Abund.	34.20	50.00	62.40	29.10	35.90	35.60	22.10	TOL	GEN	YES
(Luxilus chrysocephalus)	Population	443.00	138.00	839.00	54.00	49.00	21.00	93.00			
	95% Upper	646.38	158.90	3990.00	66.49	151.66	21.00	111.46			

Table 6.51. (continued)

Species		Site 1	2	3	4	5	6	HC3	Tolerance Classification	Trophic Group	Lithophilic Spawner?
Stoneroller	Rel. Abund.	52.50	34.50	17.40	37.60	25.60	1.70	43.70	-	HER	NO
(Campostoma anomalum)	Population	439.00	108.00	199.00	75.00	27.00	1.00	217.00			
	95% Upper	483.88	143.92	a	96.29	64.28	1.00	275.55			
Catostomidae	Rel. Abund.	0.00	0.00	0.00	1.20	2.60	0.00	1.90			
Golden redhorse	Population	b	b	b	2.00	2.00	b	7.00	MIN	BIN	YES
(Moxostoma erythrurum)	95% Upper	b	b	b	a	2.11	b	7.21			
Northern hogsucker	Rel. Abund.	0.00	0.00	0.40	8.50	6.40	5.10	6.80	MIN	BIN	YES
(Hypentilium nigricans)	Population	b	b	1.00	14.00	5.00	3.00	25.00			
	95% Upper	b	b	1.00	20.10	6.06	a	27.76			
White sucker	Rel. Abund.	1.70	1.70	2.10	0.00	0.00	0.00	0.00	TOL	GEN	YES
(Catostomus commersoni)	Population	21.00	4.00	5.00	b	b	b	b			
	95% Upper	a	4.35	11.26	b	b	b	b			
Ictaluridae	Rel. Abund.	0.40	0.00	0.00	1.80	2.60	0.00	0.00			
Yellow bullhead	Population	3.00	b	b	3.00	2.00	b	b	TOL	BIN	NO
(Ictalurus natalis)	95% Upper	6.83	b	b	3.05	3.04	b	b			
Poeciliidae	Rel. Abund.	0.70	0.80	0.80	0.00	0.00	0.00	0.00			
Mosquitofish	Population	5.00	2.00	2.00	b	b	b	b	TOL	INS	NO
(Gambusia affinis)	95% Upper	a	3.04	3.04	b	b	b	b			
Centrarchidae	Rel. Abund.	1.70	0.40	0.40	4.80	1.30	5.10	0.50			
Blugill sunfish	Population	12.00	1.00	1.00	11.00	1.00	4.00	2.00	-	INS	NO
(Lepomis macrochirus)	95% Upper	12.00	1.27	2.76	a	1.00	a	2.81			

Table 6.51. (continued)

Species		Site 1	2	3	4	5	6	HC3	Tolerance Classification	Trophic Group	Lithophilic Spawner?
Green sunfish	Rel. Abund.	0.10	0.00	0.00	0.00	1.30	0.00	0.00	TOL	INS	NO
(L. cyanellus)	Population	1.00	b	b	b	1.00	b	b			
	95% Upper	2.76	b	b	b	1.00	b	b			
Redbreast sunfish	Rel. Abund.	3.50	2.50	13.20	10.90	16.70	8.50	0.80	-	INS	NO
(L. auritus)	Population	27.00	6.00	52.00	25.00	16.00	5.00	3.00			
	95% Upper	39.29	a	122.93	76.38	a	5.23	3.00			
Rockbass	Rel. Abund.	0.00	0.00	0.00	0.60	1.30	1.70	4.40	SIN	PIS	NO
(Ambloplities rupestris)	Population	b	b	b	1.00	1.00	1.00	16.00			
	95% Upper	b	b	b	1.27	1.00	1.00	18.27			
Largemouth bass	Rel. Abund.	0.00	0.00	0.00	0.00	0.00	1.70	0.00	-	PIS	NO
(Micropterus salmoides)	Population	b	b	b	b	b	1.00	b			
	95% Upper	b	b	b	b	b	1.00	b			
Percidae	Rel. Abund.	0.00	0.00	0.00	0.00	0.00	1.70	0.00			
Logperch	Population	b	b	b	b	b	1.00	b	MIN	INS	YES
(Percina caprodes)	95% Upper	b	b	b	b	b	1.00	b			
Stripetail darter	Rel. Abund.	0.00	0.00	0.00	0.00	0.00	0.00	0.80	SIN	BIN	NO
(Etheostoma kennicotti)	Population	b	b	b	b	b	b	3.00			
	95% Upper	b	b	b	b	b	b	a			
Snubnose darter	Rel. Abund.	0.00	0.00	0.00	0.00	0.00	1.70	3.30		BIN	NO
(E. simoterum)	Population	b	b	b	b	b	1.00	12.00			
	95% Upper	b	b	b	b	b	1.00	16.18			

Table 6.51. (continued)

Species		Site 1	2	3	4	5	6	HC3	Tolerance Classification	Trophic Group	Lithophilic Spawner?
Sciaenidae	Rel. Abund.	0.00	0.00	0.00	0.00	1.30	0.00	0.00			
Freshwater drum	Population	b	b	b	b	1.00	b	b	-	BIN	NO
(Aplodinotus grunniens)	95% Upper	b	b	b	b	0.00	b	b			
Cottidae	Rel. Abund.	0.00	0.00	0.00	3.00	2.60	5.10	9.30			
Banded skulpin	Population	b	b	b	5.00	2.00	3.00	76.00	MIN	BIN	NO
(Cottus carolinae)	95% Upper	b	b	b	6.06	2.00	3.56	487.82			

MIN= MODERATELY INTOLERANT
SIN= SLIGHTLY INTOLERANT
TOL= TOLERANT
"-"= MODERATELY TOLERANT
BIN= BENTHIC INSECTIVORE
GEN= GENERALIST FEEDER
INS= INSECTIVORE
OMN= OMNIVORE
HER= HERBIVORE

a = Unable to calculate upper population estimate because number of fish captured did not decrease each pass; thus, the formula fails.
b = No individuals of this species were captured at the site.

Table 6.52. Tolerance and trophic classifications of the ten species of fish that were captured only in EFPC during the SAIC sampling in October 1991.

Species	Tolerance classification	Trophic classification
Mosquito fish (*Gambusia affinis*)	Tolerant	insectivore
Bluntnose minnow (*Pimephales notatus*)	Tolerant	omnivore
Fathead minnow (*P. promelas*)	Tolerant	omnivore
Green sunfish (*Lepomis cyanellus*)	Tolerant	insectivore
White sucker (*Catostomus commersoni*)	Tolerant	omnivore
Yellow bullhead (*Ameiurus natalis*)	Tolerant	benthic insectivore
Freshwater Drum (*Aplodinotus grunniens*)	Moderately tolerant	benthic insectivore
Gizzard Shad (*Dorosoma cepedianum*)	Moderately intolerant	omnivore
Largemouth Bass (*Micropterus salmoides*)	Moderately intolerant	piscivore
Logperch (*Percina caprodes*)	Moderately intolerant	insectivore

Tolerant fish species are defined as being better adapted to survive in various conditions of degraded water quality, including both physical (i.e., habitat) and chemical degradation (including toxicant exposure) (Plafkin et al. 1989; Ohio EPA 1988). The Ohio EPA classification of fish tolerances was based on a large data base of fish catch data from ~2000 sites, and also incorporated results from laboratory bioassays, historical distribution records, and "best professional judgment" (Ohio EPA 1988). A fish community dominated by tolerant species indicates degraded environmental conditions.

Tolerant fish species comprised nearly 75% of the species captured at Site 1, but steadily decreased downstream to 17% of the species captured at Site 6 (Fig. 6.58). Of the species captured at the reference site, 29% were tolerant species. The decrease in percentage of tolerant species downstream from the Y-12 Plant suggests a gradual improvement in water quality with increased distance from the plant. However, although the percentage of tolerant species captured at Sites 5 and 6 in EFPC was less than at the reference site, the relative abundance of all tolerant species at the reference site combined was still less than at any of the EFPC sites (Fig. 6.59). This observation, coupled with the fact that 7 of the 10 fish species that were captured only in EFPC were tolerants, indicates that fish of all sampling sites in EFPC are negatively impacted compared to fish at the Hinds Creek reference stream.

Striped shiners and stonerollers were the dominant species at every site except Site 6 (Fig. 6.60); their combined relative abundances ranged from 64% to 94%. Striped shiners and bluntnose minnows were the two dominant species at Site 6, having combined relative abundances of 55%. The relative abundance of redbreast sunfish generally increased downstream from Site 1, but decreased at Site 6 and was very low at the reference site (< 1%). The low abundance of redbreast sunfish at the reference site was most likely an artifact of the single-event sampling at Hinds Creek. Researchers conducting the bioaccumulation study component of the Y-12 BMAP have reported occasional low abundances of rebreast sunfish in Hinds Creek (George Southworth, Appendix R).

The BMAP studies (Hinzman 1992) reported more total species in EFPC (i.e., 30 versus 22) and generally more species per site than in this study. However, the BMAP sampled biannually from 1986 to 1988, whereas this study sampled once in the fall of 1991. The increased number of samples over a longer period of record in the BMAP investigations could have resulted in the larger number of observed species.

Trends for species richness and composition in this study were similar to historical results for EFPC reported during the BMAP (Hinzman 1992). Both studies reported that species

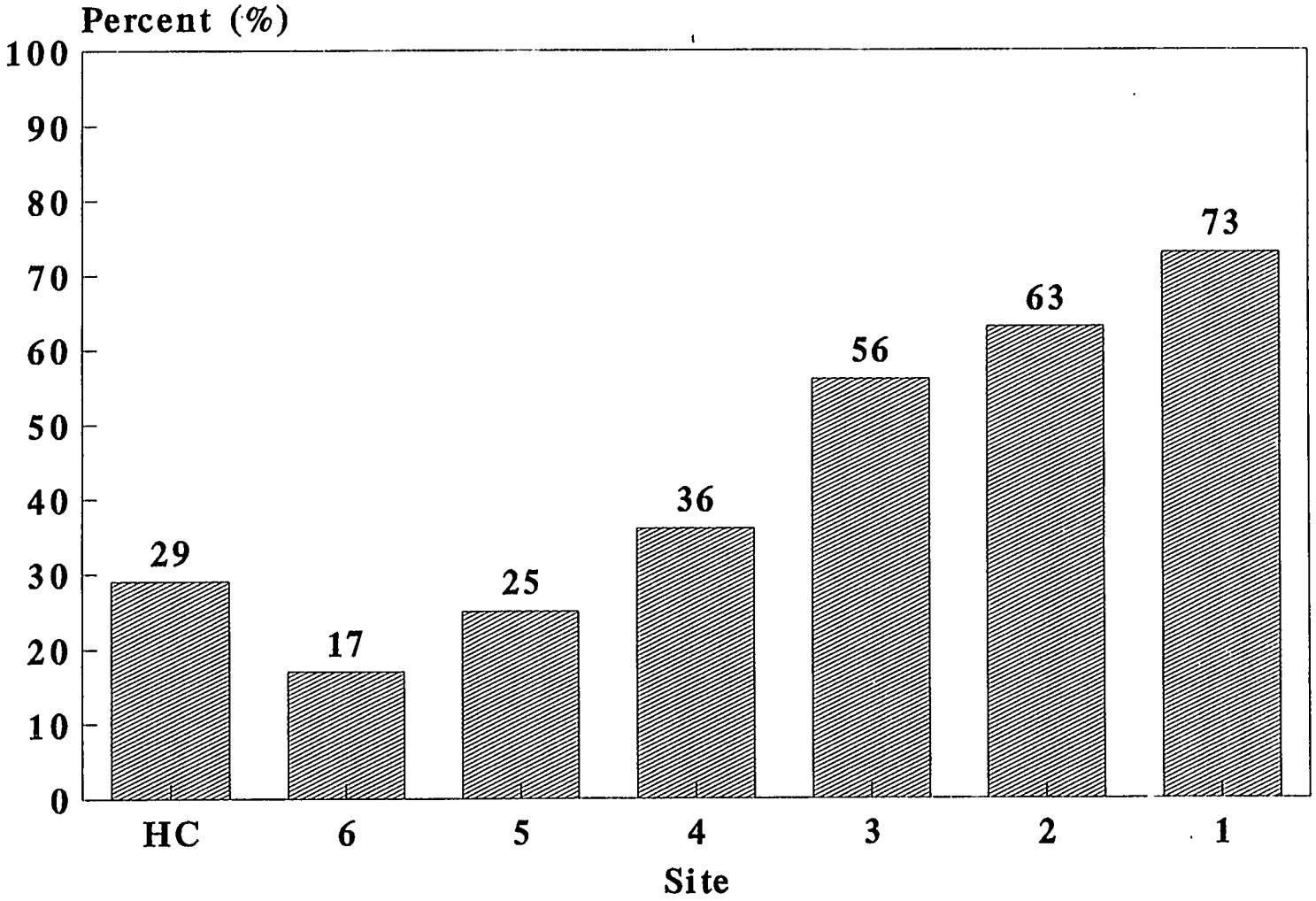

Fig. 6.58. Percentage of captured fish species from EFPC and Hinds Creek classified as tolerant of degraded water quality, October 7–12, 1991.

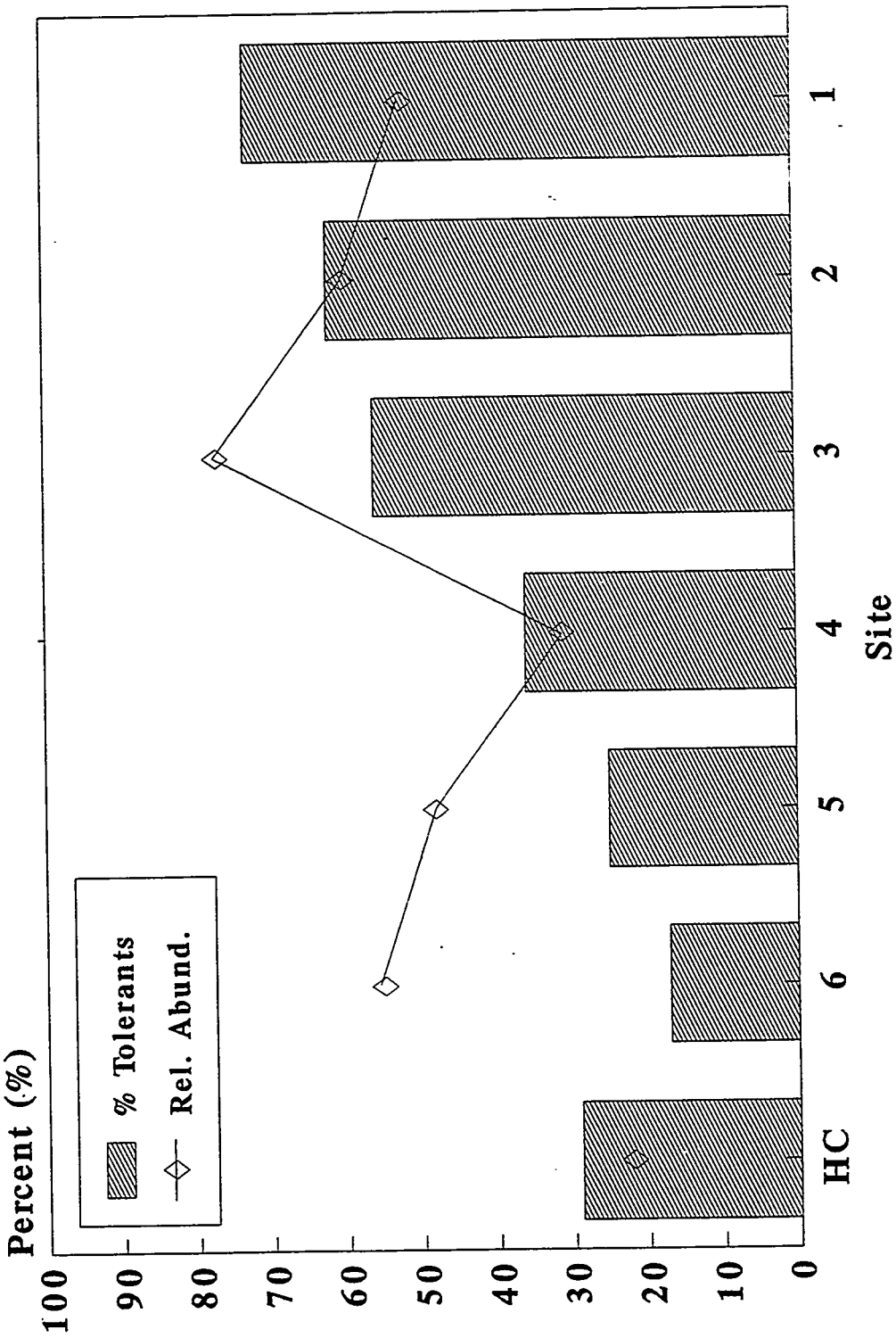

Fig. 6.59. Percentage of captured fish species from EFPC and Hinds Creek classified as tolerant of degraded water quality, and the combined relative abundance of all tolerant individuals.

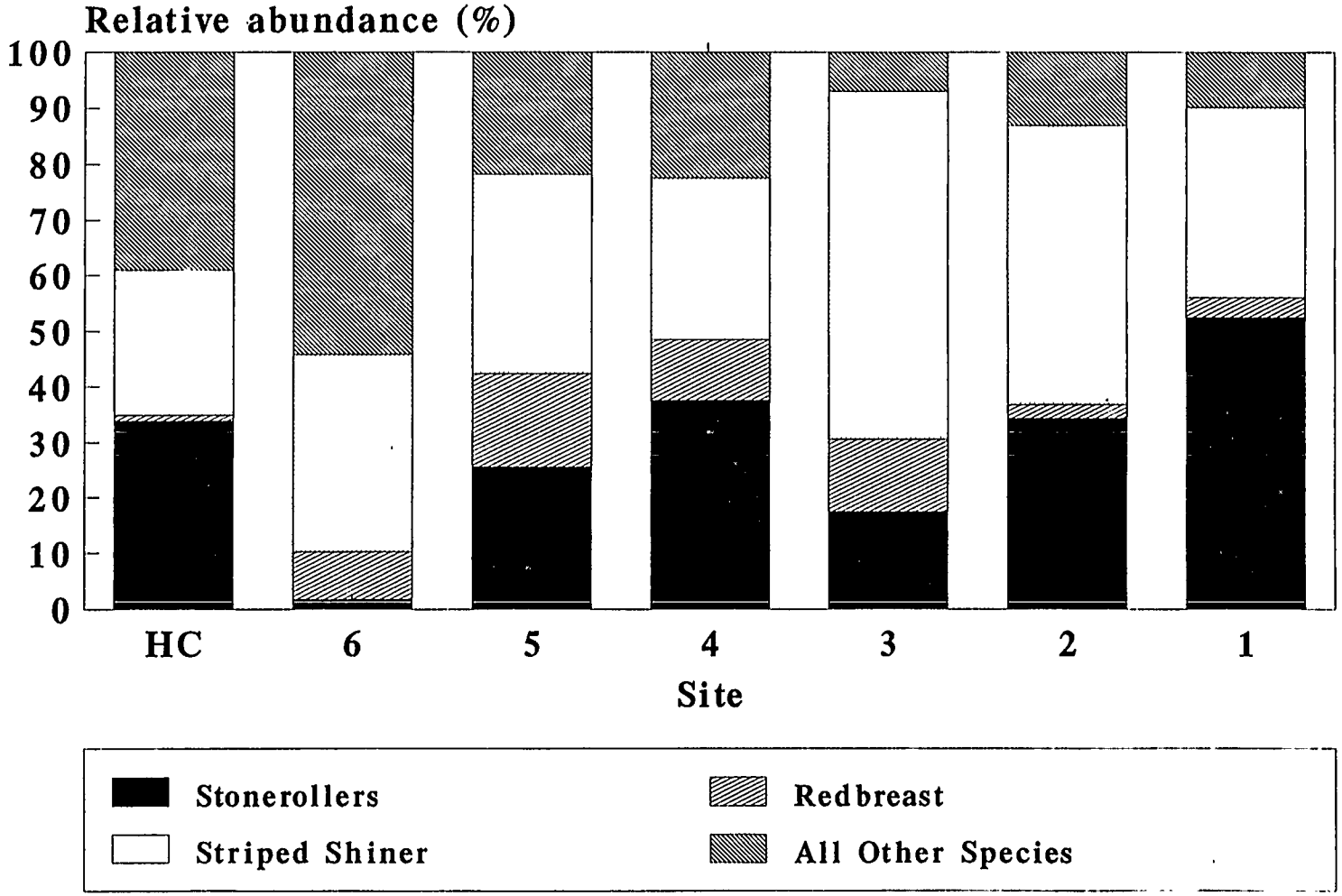

Fig. 6.60. Relative abundance of redbreast sunfish, stonerollers, striped shiners, and the collective remaining fish species captured at each site in EFPC and Hinds Creek.

richness generally increased downstream from the Y-12 Plant. In both studies stonerollers and striped shiners were the dominant species.

Abundance (density). Fish population density varied considerably among the six EFPC sites (Fig. 6.61). Sites 3 and 1 had the greatest estimated fish densities. Both sites had over 16 fish per 10 m^2, which was nearly double the reference site fish density of 9.34 fish per 10 m^2. Site 2, located between Sites 1 and 3, yielded an estimate of only 4.35 fish per 10 m^2. The remaining downstream sites in EFPC (from Site 4 to Site 6) had decreasing population density estimates, ranging from 2.78 to 0.43 fish/10 m^2.

Total fish densities in this study were considerably less than those reported during the BMAP investigations from 1985 through 1988 (Loar 1992; Hinzman 1992). However, a general trend of decreasing fish density with increasing stream size and downstream distance from the Y-12 Plant was observed in both this study and the second BMAP study.

Diversity. Species diversity, expressed as the Shannon-Wiener Diversity Index, progressively increased downstream from Site 1, except for a decrease at Site 3 (Fig. 6.62). Only the EFPC site farthest downstream had a greater species diversity index than the reference site. The three EFPC sites nearest the Y-12 Plant, at which the greatest surface water and floodplain soil mercury concentrations were obtained, had the lowest diversity indices.

A measure of evenness of species abundances (J) that corrects for species richness is calculated as

$$J = H'/H'_{max}$$

where H'_{max} = log n, and n = number of species. By definition, maximum species diversity (H'_{max}) for a given number of species would occur if all of the species were equally abundant. An evenness value of 1.00 would thus indicate an equal abundance of individuals among all species and maximum species diversity for the given number of species. The evenness was < 0.50 at Site 1 and, except for Site 3, progressively increased downstream to nearly 0.80 at Site 6 (Fig. 6.63). Evenness at the reference site was 0.67, which was greater than at the three sites closest to the Y-12 Plant, but slightly less than at the three remaining EFPC sites downstream. The low evenness values from Site 1 to Site 3 in EFPC indicate that fish communities at those sites are dominated by a few species.

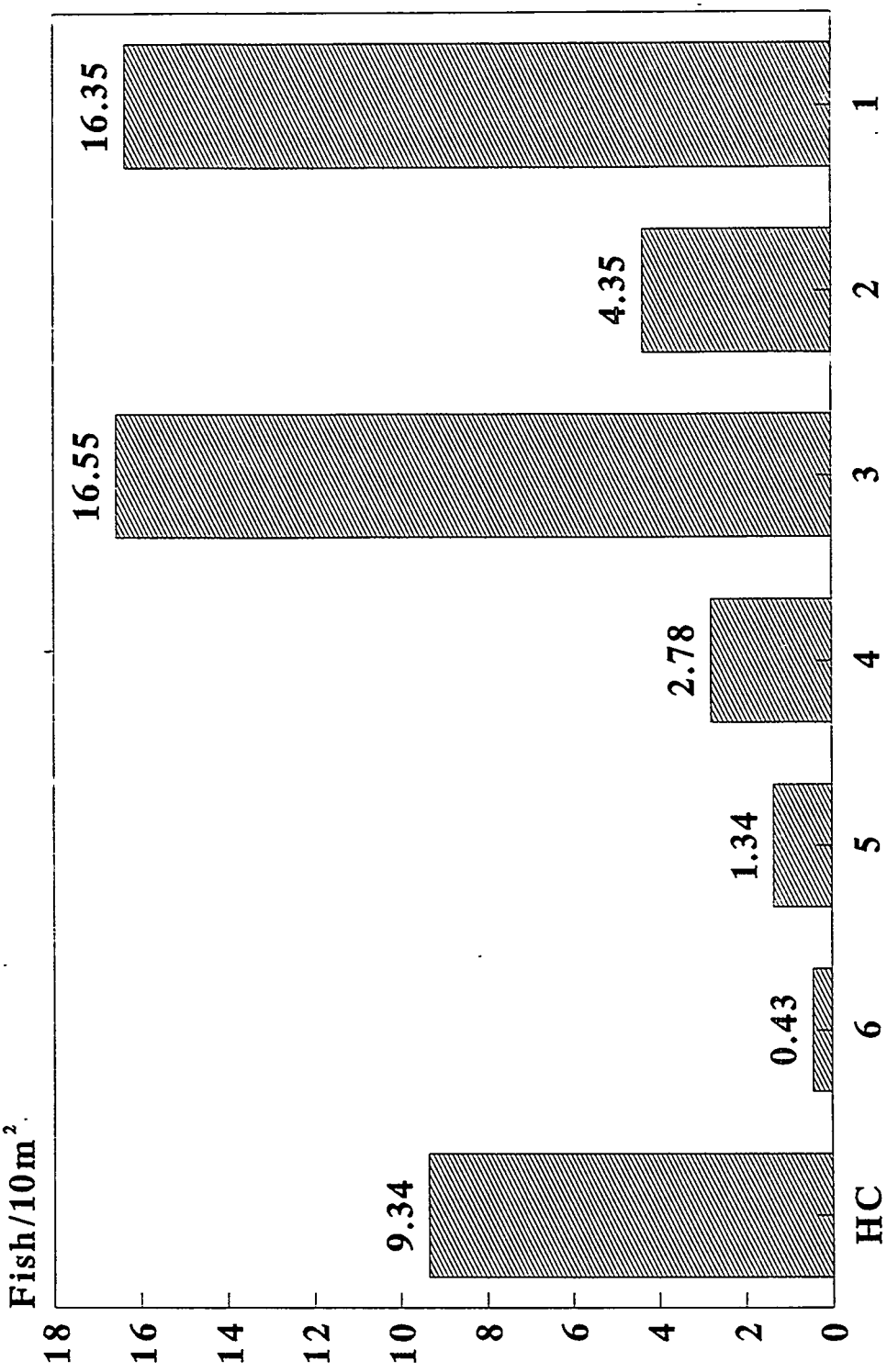

Fig. 6.61. Fish density, expressed as total number of fish per 10 m², at sites in EFPC and Hinds Creek, October 7–12, 1991.

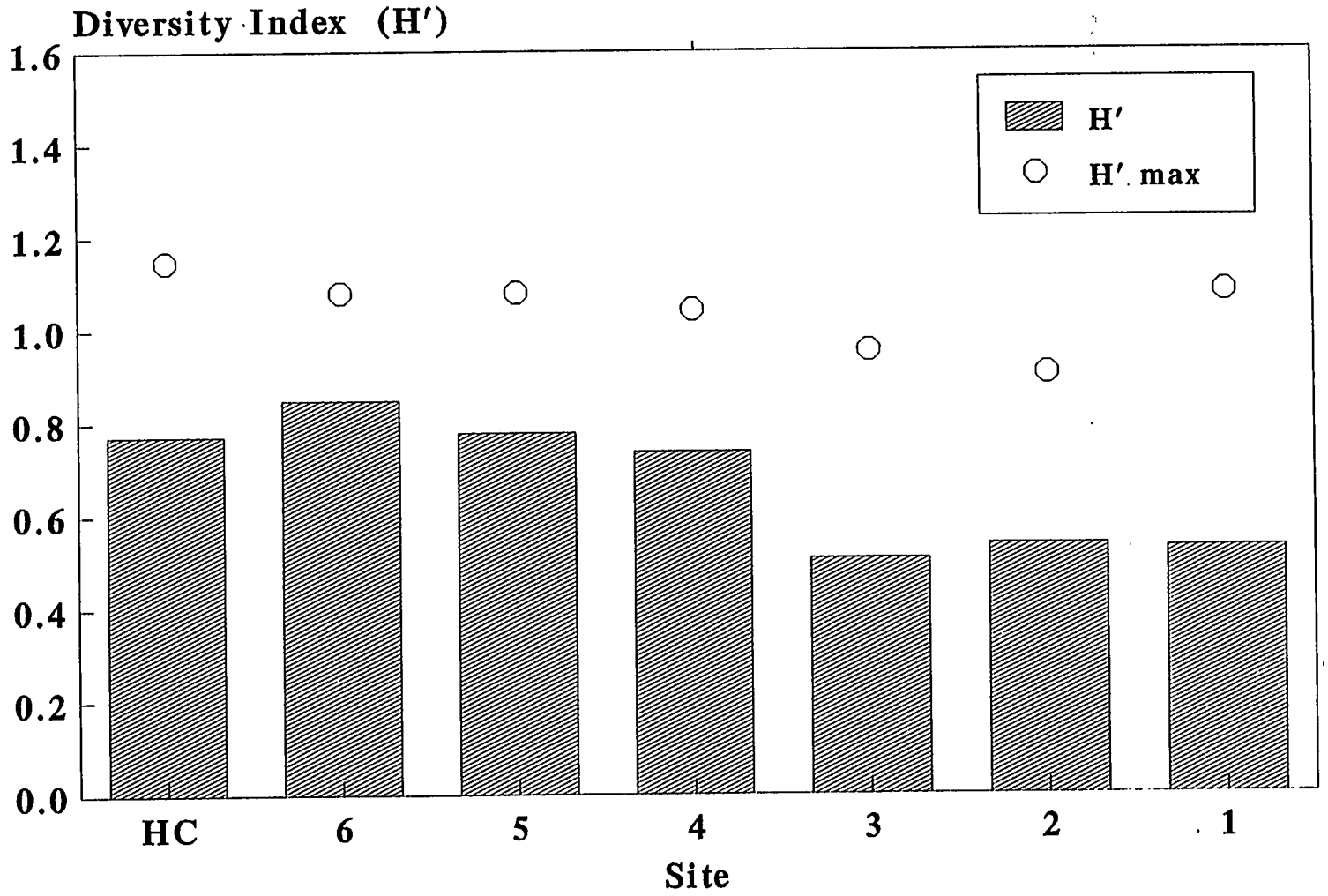

Fig 6.62. Observed fish species diversity (H') and maximum possible fish diversity (H'_{max}) at sites in EFPC and Hinds Creek, October 7–12, 1991.

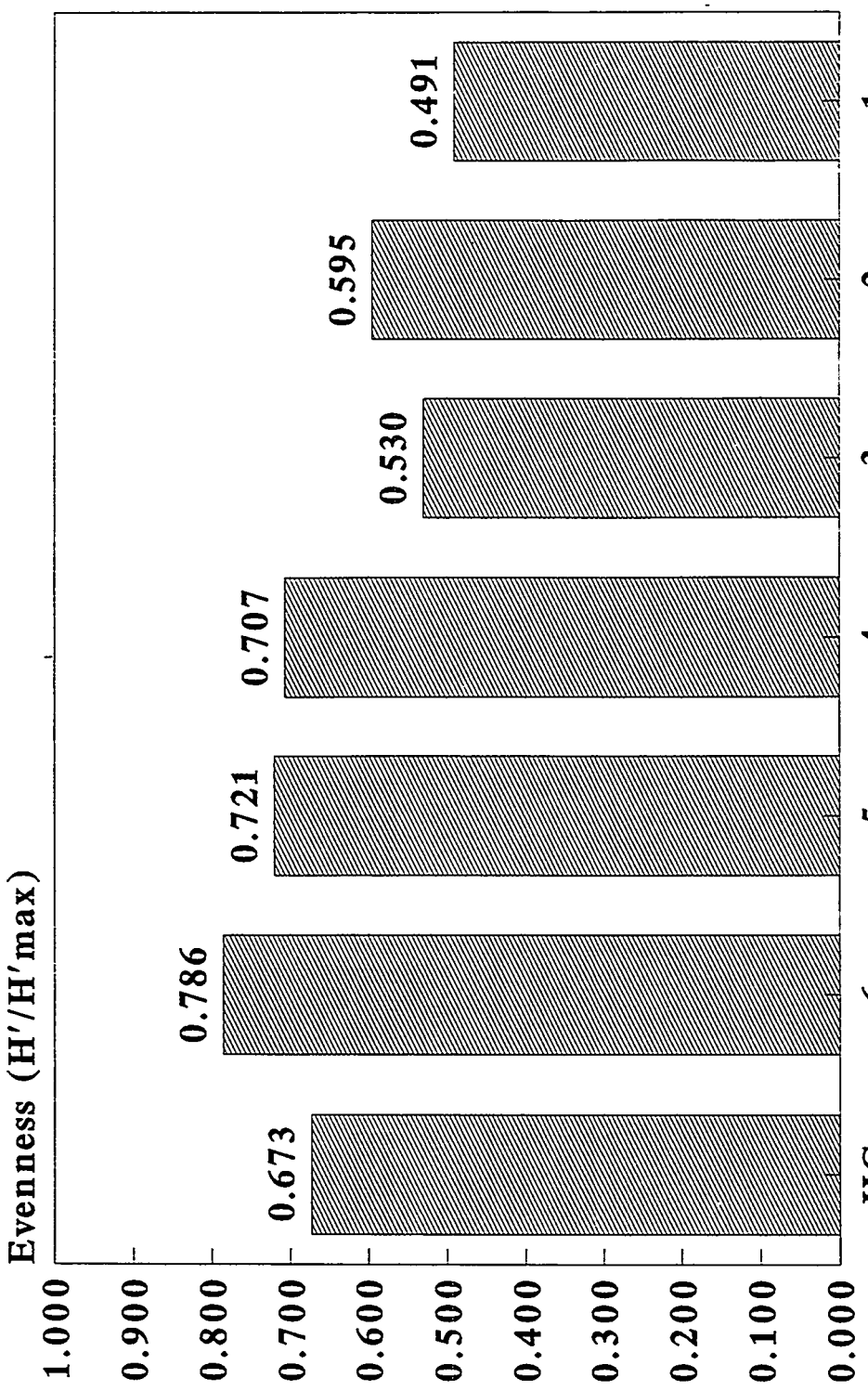

Fig. 6.63. Species evenness (H'/H'_{max}) for fish collected from EFPC and Hinds Creek, October 7-12, 1991

Limitations and uncertainties

The greatest limitation on the interpretation of any of these aquatic biota sampling data is that they represent only a single point in time. Time and funding limitations restricted the experimental design to unreplicated surveys that could be completed in a matter of days. Single-sampling designs do not permit observation of the seasonal variations that are expected to occur in EFPC and Hinds Creek. In addition, trends cannot be detected unless comparisons can be made to historical data.

An important source of uncertainty in the fish data is the backpack electrofishing method. The efficiency of backpack electrofishing is dependent on a number of factors, including stream size and depth, operating voltage, water conductivity, number and types of hiding refuges for fish, fish size, water clarity and flow, operator performance, and thoroughness of retrieval of stunned fish. Although blocking nets were placed across the upper and lower boundaries of the sampling reach to minimize immigration and emigration of fish, some degree of uncertainty exists as to whether all individuals have been captured.

A third source of uncertainty concerning only population density and relative abundance data is the methods of Carle and Strub (1978). However, the uncertainty in population estimates is quantified because they are expressed with 95% confidence limits.

6.3.3.2 Benthic macroinvertebrate survey

Quantitative benthic macroinvertebrate samples were collected from riffle habitats at six sites in EFPC and one at the Hinds Creek reference site during October 22 through 29, 1991, using a Surber sampler according to technical procedure *TP-309-7: Benthic Macroinvertebrate Sampling (Lotic Habitats)* (LWA 1990). The riffle locations were all within the stream segments sampled for the fish population surveys described in Sect. 6.3.3.1. The purpose of benthic macroinvertebrate sampling was to obtain data on taxa richness and composition, density, and diversity and, if possible, to collect a sufficiently large composite sample to analyze for contaminants of concern.

Sampling methods

Surber samples (0.1 m^2) were collected systematically, beginning at the downstream end of the riffle and continuing along a line perpendicular to the stream banks to the opposite lateral edge of the riffle. Sampling then progressed upstream in increments of ~0.05 m and continued across the riffle to the lateral edge. This zigzag pattern was repeated until the entire riffle was

sampled or until sufficient numbers of organisms were obtained for the population and body burden analyses. The number of Surber samples collected at each site ranged from 7 to 25; the total area sampled at each site thus ranged from 0.7 to 2.5 m^2 (Table 6.53). The combined samples were washed into white plastic trays to assist in locating and removing any organisms. Organisms clinging to the net were also removed with stainless steel forceps. All organisms for the population characterization were placed into labeled jars containing 80% ethanol. Any organisms for contaminant body burden analysis were collected using decontaminated equipment and placed into a Whirl-pac™ plastic bag kept in crushed ice. The plastic bag was then placed into a labeled, chemically clean glass jar, sealed with custody tape, and kept in a cooler filled with ice until being returned from the field and locked in a freezer.

Density estimates for each site are expressed as number of organisms per m^2 and were calculated based on the total number of organisms captured per total area sampled at each site.

Diversity (H′) was calculated using the Shannon-Wiener Index:

$$H' = -\sum_{i=1}^{n} p_i * \log_2 p_i$$

where p_i equals the proportion of family i in the sample. The binary log is used to facilitate comparison with BMAP historical results. Maximum diversity (H'_{max}) was calculated for each site by setting each p_i equal to $1/n$, where n=total number of families at that site. Evenness (J) was calculated as H'/H'_{max}.

During the fish sampling conducted two weeks prior to the benthic macroinvertebrate sampling, stream morphological, physical, and chemical conditions were characterized. These parameters were not expected to change appreciably in the relatively short time between the two sampling periods and thus were not repeated.

Results

Taxa richness. Twenty-two different families of benthic macroinvertebrates were collected from the six sites in EFPC, compared with fifteen families from the single site in Hinds Creek. Table 6.54 summarizes the taxonomic and functional feeding group classifications, number of individuals per family, and relative abundances of benthic macroinvertebrates captured in EFPC and in Hinds Creek [functional feeding group classifications for the aquatic insects were based

Table 6.53. Summary of benthic macroinvertebrate sampling at sites in EFPC and Hinds Creek, October 22 - 29, 1991.

Parameter	Site						
	1	2	3	4	5	6	HC3
Surber replicates	18	24	20	25	12	20	7
Area (in square meters)	1.80	2.40	2.00	2.50	1.20	2.00	0.70
Total number of individuals collected	276	813	139	183	226	133	130
Individuals per square meters	153	339	70	73	188	67	186
Total richness	7	10	10	14	9	12	15
EPT richness	1	4	3	4	4	3	7

Table 6.54. Taxonomic classifications, functional feeding group classifications, tolerance values, number of individuals, and relative abundance of benthic macroinvertebrates in EFPC and Hinds Creek, October 22 - 29, 1991

Class	Order	Family	Functional feeding group	Tolerance Values*	Number of individuals per family	Relative Abundance (%)
		Site 1				
Crustacea	Decapoda	Astacidae	Scav/Pred	6	2	0.72
Insecta	Odonata	Coenagrionidae**	Pred	9	6	2.17
Insecta	Megaloptera	Corydalidae	Pred	0	4	1.45
Insecta	Trichoptera	Hydropsychidae	Filcol	4	231	83.70
Insecta	Coleoptera	Elmidae	Scrap	4	3	1.09
Insecta	Diptera	Chironomidae	Filcol/pred	6-8	2	0.72
Pelecypoda	Bivalvia	Corbiculidae**	Filcol	NA	28	10.14
			TOTALS		276	100
		Site 2				
Crustacea	Decapoda	Astacidae	Scav/Pred	6	1	0.12
Insecta	Ephemeroptera	Baetidae	Scrap	4	1	0.12
Insecta	Ephemeroptera	Oligonneuriidae	Filcol	2	1	0.12
Insecta	Megaloptera	Corydalidae	Pred	0	2	0.25
Insecta	Trichoptera	Hydropsychidae	Filcol	4	600	73.80
Insecta	Trichoptera	Philopotamidae	Filcol	3	1	0.12
Insecta	Coleoptera	Elmidae	Scrap	4	10	1.23
Insecta	Diptera	Tipulidae	Shred	3	2	0.25
Pelecypoda	Bivalvia	Corbiculidae	Filcol	NA	194	23.86
Gastropoda	Basommatophora	Ancylidae	Grazer	6	1	0.12
			TOTALS		813	100
		Site 3				
Crustacea	Decapoda	Astacidae	Scav/Pred	6	4	2.88
Insecta	Ephemeroptera	Baetidae	Scrap	4	5	3.60
Insecta	Ephemeroptera	Heptageniidae	Scrap	4	98	70.50
Insecta	Odonata	Coenagrionidae	Pred	9	1	0.72
Insecta	Megaloptera	Corydalidae	Pred	0	1	0.72
Insecta	Trichoptera	Hydropsychidae	Filcol	4	3	2.16
Insecta	Coleoptera	Elmidae	Scrap	4	18	12.95
Insecta	Diptera	Chironomidae	Filcol/Pred	6-8	1	0.72
Insecta	Diptera	Tabanidae**	Scrap	6	1	0.72
Pelecypoda	Bivalvia	Corbiculidae	Filcol	NA	7	5.04
			TOTALS		139	100

Table 6.54. (continued)

Class	Order	Family	Functional feeding group	Tolerance Values*	Number of individuals per family	Relative Abundance (%)
		Site 4				
Crustacea	Isopoda	Asellidae**		8	8	4.37
Insecta	Ephemeroptera	Baetidae	Scrap	4	9	4.92
Insecta	Ephemeroptera	Heptageniidae	Scrap	4	22	12.02
Insecta	Ephemeroptera	Tricorythidae**	Colgat	4	3	1.64
Insecta	Odonata	Coenagrionidae	Pred	9	2	1.09
Insecta	Odonata	Gomphidae**	Pred	1	1	0.55
Insecta	Trichoptera	Hydropsychidae	Filcol	4	105	57.38
Insecta	Coleoptera	Elmidae	Scrap	4	7	3.83
Insecta	Coleoptera	Psephenidae**	Scrap	4	1	0.55
Insecta	Diptera	Simulidae	Filcol	6	1	0.55
Insecta	Diptera	Tipulidae	Shred	3	1	0.55
Pelecypoda	Bivalvia	Corbiculidae	Filcol	NA	17	9.29
Gastropoda	Basommatophora	Ancylidae	Grazer	6	5	2.73
Gastropoda	Basommatophora	Physidae**	Grazer	8	1	0.55
			TOTALS		183	100
		Site 5				
Insecta	Ephemeroptera	Baetidae	Scrap	4	10	4.42
Insecta	Ephemeroptera	Heptageniidae	Scrap	4	78	34.51
Insecta	Ephemeroptera	Tricorythidae	Colgat	4	20	8.85
Insecta	Trichoptera	Hydropsychidae	Filcol	4	91	40.27
Insecta	Coleoptera	Elmidae	Scrap	4	1	0.44
Insecta	Diptera	Chironomidae	Filcol/Pred	6-8	8	3.54
Insecta	Diptera	Simuliiade	Filcol	6	5	2.21
Pelecypoda	Bivalvia	Corbiculidae	Filcol	NA	9	3.98
Gastropoda	Basommatophora	Ancylidae	Grazer	6	4	1.77
			TOTALS		226	100
		Site 6				
Crustacea	Isopoda	Asellidae		8	3	2.26
Insecta	Ephemeroptera	Heptageniidae	Scrap	4	4	3.01
Insecta	Ephemeroptera	Tricorythidae	Colgat	4	64	48.12
Insecta	Trichoptera	Hydropsychidae	Filcol	4	1	0.75
Insecta	Coleoptera	Elmidae	Scrap	4	1	0.75
Insecta	Coleoptera	Psephenidae	Scrap	4	1	0.75
Insecta	Diptera	Chironomidae	Filcol/Pred	6-8	11	8.27
Insecta	Diptera	Tipulidae	Shred	3	1	0.75
Pelecypoda	Bivalvia	Corbiculidae	Filcol	NA	40	30.08
Gastropoda	Basommatophora	Ancylidae	Grazer	6	1	0.75
Gastropoda	Basommatophora	Planorbidae**		6	1	0.75
Gastropoda	Mesogastropoda	Pleuroceridae	Grazer	NA	5	3.76
			TOTALS		133	100

Table 6.54. (continued)

Class	Order	Family	Functional feeding group	Tolerance Values*	Number of individuals per family	Relative Abundance (%)
		Site HC3				
Crustacea	Decapoda	Astacidae	Scav/Pred	6		0.00
Insecta	Ephemeroptera	Baetidae	Scrap	4	12	9.23
Insecta	Ephemeroptera	Heptageniidae	Scrap	4	26	20.00
Insecta	Ephemeroptera	Oligoneuriidae	Filcol	2	6	4.62
Insecta	Megaloptera	Corydalidae	Pred	0	1	0.77
Insecta	Plecoptera	Taeniopterygidae	Shred/Scrap	2	1	0.77
Insecta	Trichoptera	Hydropsychidae	Filcol	4	32	24.62
Insecta	Trichoptera	Limnephilidae	Shred	4	1	0.77
Insecta	Trichoptera	Philopotamidae	Filcol	3	25	19.23
Insecta	Coleoptera	Elmidae	Scrap	4	5	3.85
Insecta	Diptera	Chironomidae	Filcol/Pred	6-8	8	6.15
Insecta	Diptera	Simuliidae	Filcol	6	3	2.31
Insecta	Diptera	Tipulidae	Shred	3	2	1.54
Gastropoda	Basommatophora	Ancylidae	Grazer	6	2	1.54
Gastropoda	Basommatophora	Pleuraceridae	Grazer	NA	4	3.08
			TOTALS		130	100

Scav=scavenger
Pred= predator
Filcol= filterer collector
Scrap= scraper
Colgat= collector-gatherer
Shred= shredder

* Tolerance values from Hilsenhoff [1988 (for arthropods)] and Bode [1988 (for non-arthropods)], as presented in EPA (1989), where 0=most intolerant and 10=most tolerant of pollutents.
**Family not observed at the Hinds Creek reference site.
NA=Tolerance value not available

on the designations of Merritt and Cummins (1984)]. Oligochaete worms were also collected in EFPC, but were not enumerated for reasons discussed in the "Limitations and Uncertainties" section that follows. The majority of families collected from EFPC (at 15 of 22) as well as from Hinds Creek (at 12 of 15) were insects. Two families collected at the reference site, Taeniopterygidae (Plecoptera) and Limnephilidae (Trichoptera), were not found at any of the EFPC sites. Nine families found in EFPC were not collected at the reference site (Table 6.54). Five of the families had tolerance values of 6-9 (where 10 = most tolerant, and 0 = most intolerant of pollutants), whereas 3 of the families had tolerance values of 1 or 4. The larger number of families collected from EFPC is probably attributable to the fact that six sites were sampled in EFPC, compared to only one site in Hind's Creek.

The number of families collected at each of the 6 EFPC sites, ranging from 7 to 14, was always less than the number collected at the reference site, ranging from 7 to 14 (Fig. 6.64). Total family richness was lowest at Site 1 (at 54% less than at the reference site), increased downstream to Site 4, decreased to 40% of the reference site total at Site 5, and increased to 80% of the reference site total at Site 6.

The Y-12 Plant BMAP studies (Hinzman 1992) reported 19 more families in EFPC than were observed in this study. However, the BMAP study sampled monthly from June 1986 through May 1987, whereas this study sampled once in the fall of 1991. The increased number of samples that were collected over a longer period of record in the BMAP investigations probably accounts for the larger number of observed families.

Although more families were captured during the Y-12 Plant BMAP investigations than during this investigation, three trends were observed in both studies. First, total richness was less at all EFPC sites than at the respective reference sites (i.e., Brushy Fork for the BMAP studies and Hinds Creek for this study). Second, total richness was lowest at the sites nearest the Y-12 Plant. Finally, total richness increased downstream from the Y-12 Plant, reaching a maximum at EFK 13.8 in the BMAP studies (Hinzman 1992) and at Site 4 (EFK 10.8) in this study, then decreased slightly further downstream.

The number of mayfly (Ephemeroptera), stonefly (Plecoptera), and caddisfly (Trichoptera) insect families (EPT families) were summed to determine total EPT richness, which is a biotic indicator of water quality. Although some families from these three orders are tolerant of degraded water quality conditions (including chemical pollutants), most are sensitive to degraded water quality conditions, especially families in the order Plecoptera. Total EPT richness is, therefore, expected to be lower in degraded streams.

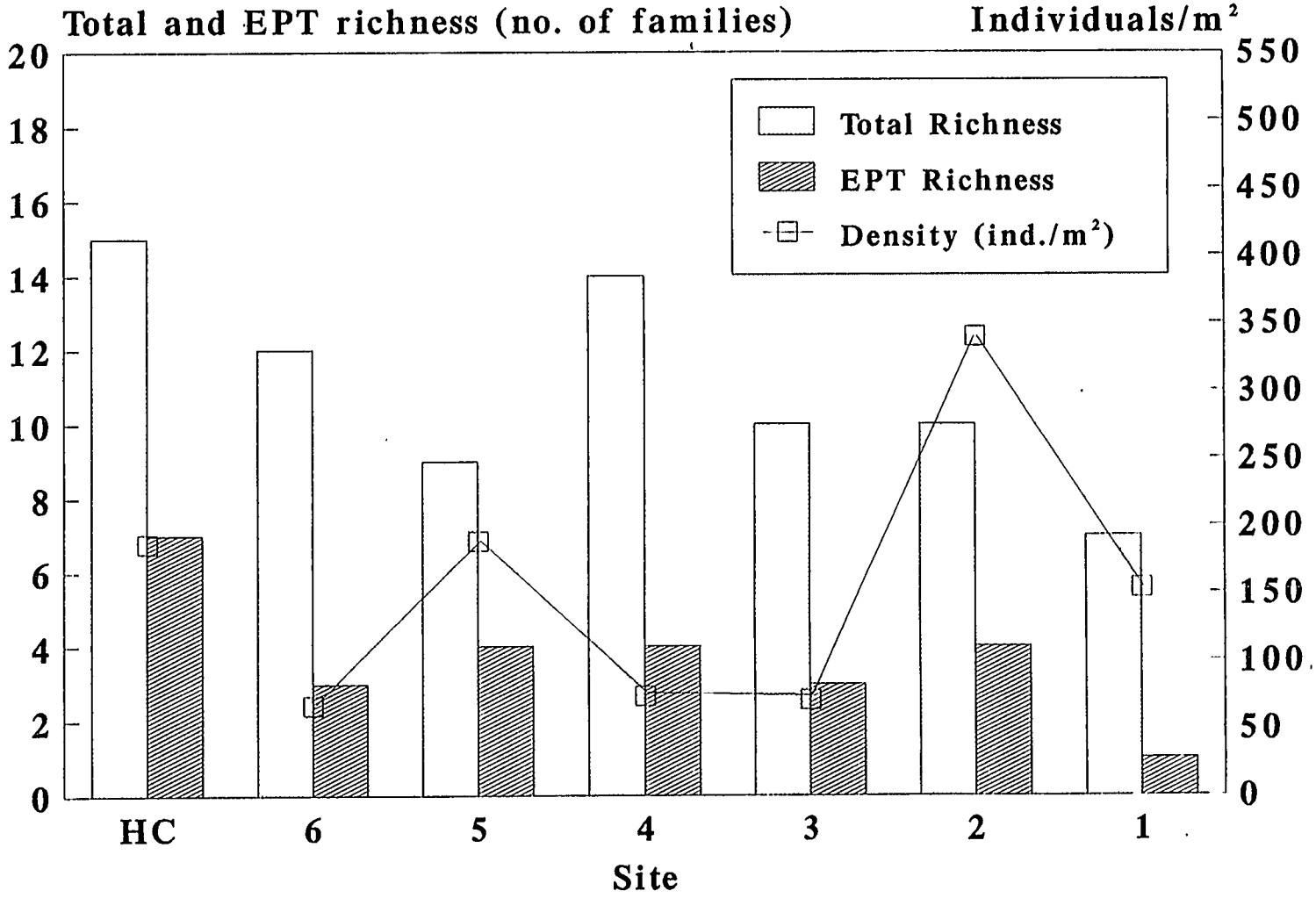

Fig. 6.64. Total family richness, EPT richness, and density (per m^2) of benthic macroinvertebrates collected from EFPC and Hinds Creek, October 22–29, 1991.

Total EPT richness was lower in all six EFPC sites than in the reference site (Fig. 6.64). The lowest total EPT richness in EFPC was observed at Site 1, with only one family present. The remaining sites in EFPC each hosted three or four EPT families, compared with seven found at the reference site. Sites in EFPC not only contained fewer families than the reference site but also had a larger proportion of non-EPT (i.e., tolerant) families (Fig. 6.64).

Some aspects of total EPT richness in this study were similar to those reported in the Y-12 Plant BMAP investigations, whereas others were dissimilar (Loar 1992; Hinzman 1992). For example, in all studies, total EPT richness was lower at every site in EFPC than at the reference site. Also, total EPT richness was lowest at the site nearest the Y-12 Plant during all three investigations. However, the longitudinal pattern of total EPT richness downstream from the Y-12 Plant was dissimilar, between the SAIC and BMAP studies. The BMAP studies reported that the longitudinal trend for total EPT richness was similar to the trend for total taxa richness, generally increasing downstream to EFK 13.8, then decreasing downstream to EFK 6.3 (Hinzman 1992). However, total EPT richness in this study increased from Site 1 to Site 2, then remained nearly constant for the remainder of the downstream EFPC sites. The pattern for total EPT richness in this investigation suggests that all of the sample sites in EFPC have degraded conditions in relation to the reference site and that Site 1 is the most degraded.

In this study, the dominant invertebrate families were families from either Trichoptera, Ephemeroptera, or, at Site 6 only, Pelecypoda (Fig. 6.65). Hydropsychidae (Trichoptera) was the dominant family at Sites 1, 2, 4, and 5 and at Hinds Creek, ranging from ~25% to 84% of individuals (Fig. 6.65). Heptageniidae (Ephemeroptera) was the most dominant family at Site 3, at 70%, and was also a co-dominant at Sites 4 and 5 and at Hinds Creek. Tricorythidae (Trichoptera) and Corbiculidae (Pelecypoda) were the dominant families at Site 6, having combined relative abundances totaling 78%.

All of the sites sampled in EFPC have benthic communities dominated by one or two families. According to Plafkin et al. (1989), a benthic community dominated by relatively few families indicates environmental stress.

The relative abundance patterns of functional feeding groups (Fig. 6.66) coincided closely with the relative abundances of dominant taxa (Fig. 6.65). The large proportions of filterer-collectors at Sites 1, 2, 4, and 5 and at Hinds Creek are a result of dominance by Hydropsychidae (Trichoptera). The large number of Hydropsychidae at these sites in EFPC indicates an abundance of suspended fine organic particulate matter, which would likely occur below the Y-12 Plant and the Oak Ridge Sewage Treatment Plant. The large relative abundance

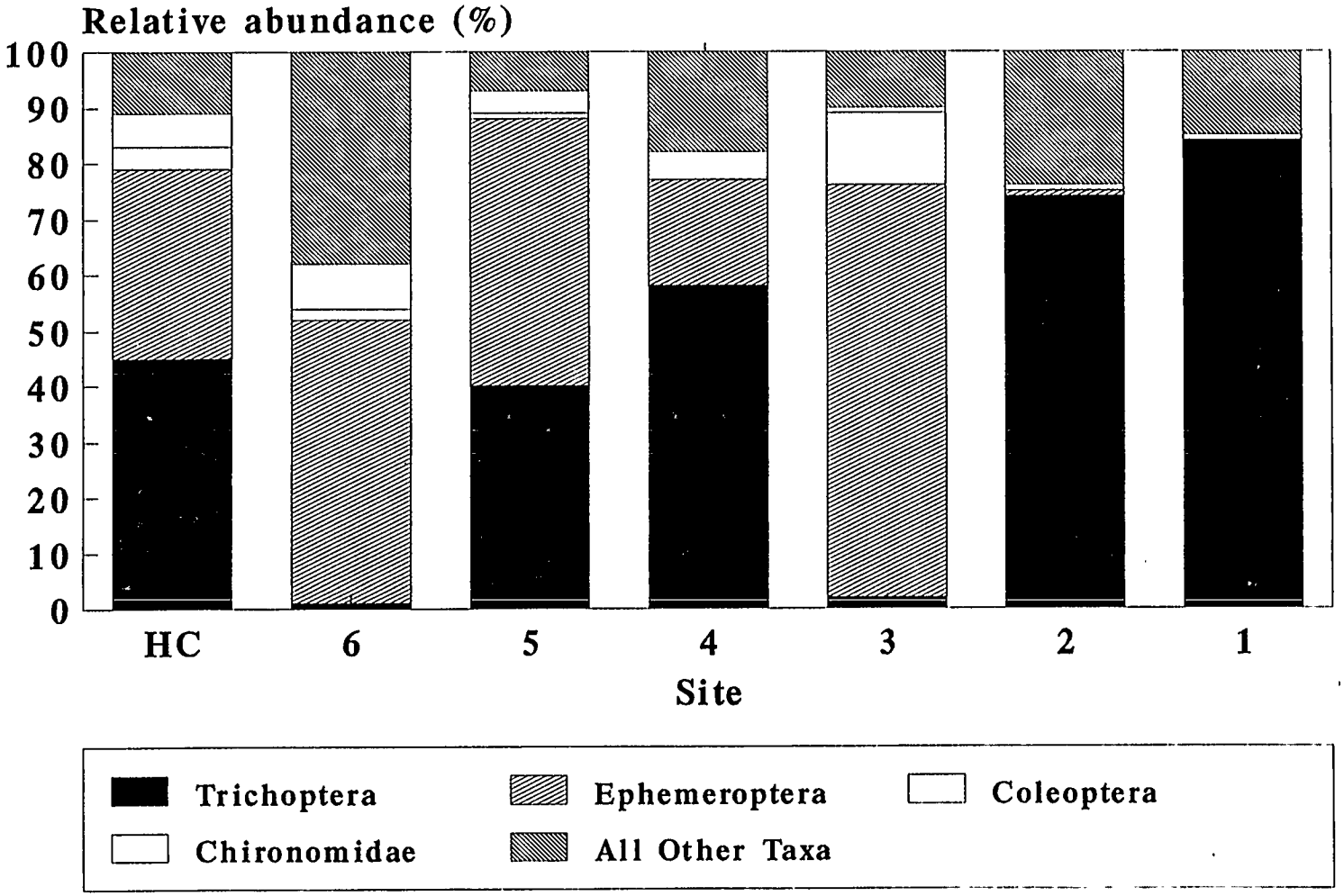

Fig. 6.65. Relative abundance of the dominant benthic macroinvertebrate taxa collected in EFPC and Hinds Creek, October 22–29, 1991.

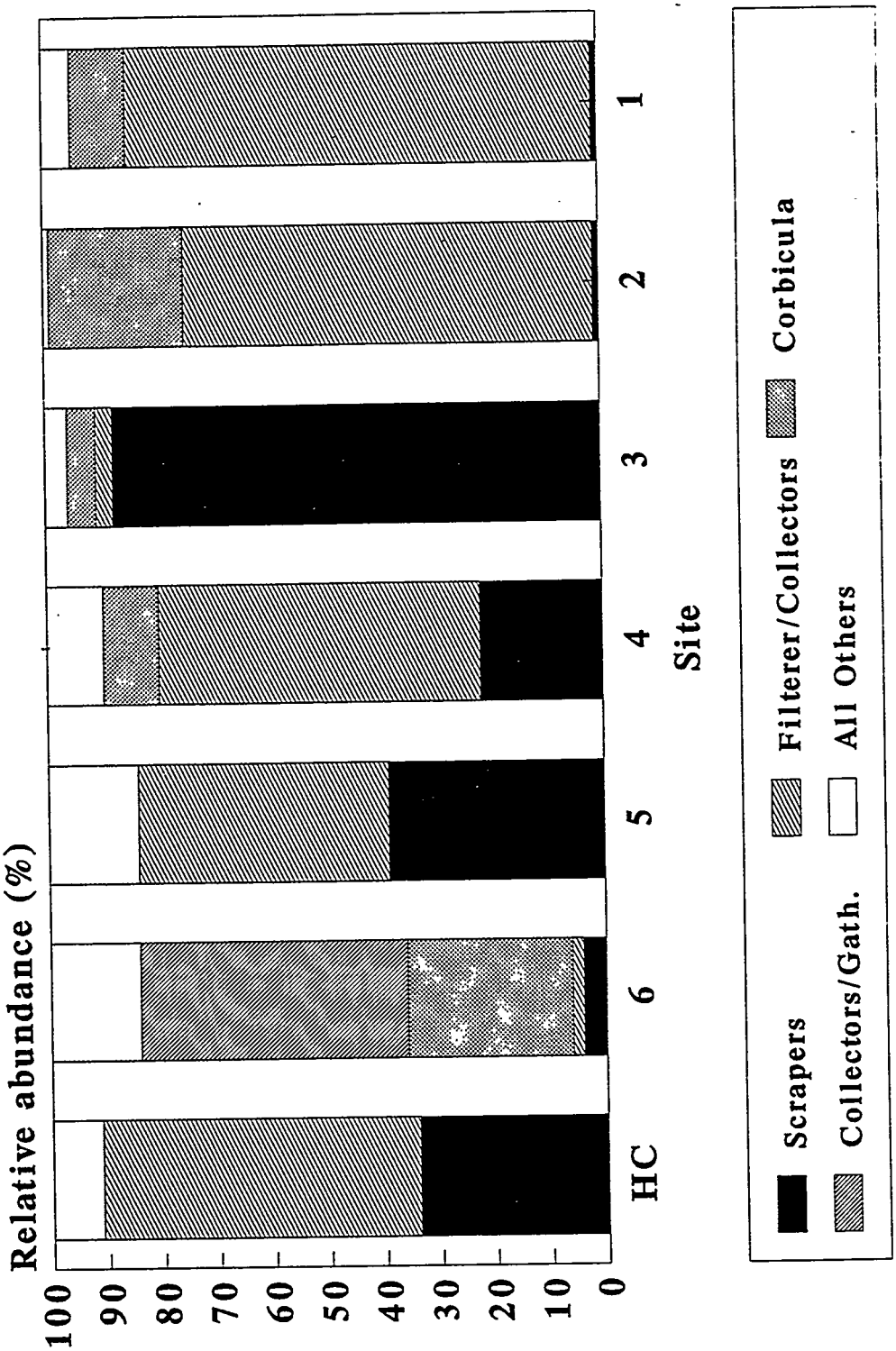

Fig. 6.66. Relative abundance of the benthic macroinvertebrate functional feeding groups collected from EFPC and Hinds Creek, October 22-29, 1991. ("All others" includes predators, grazers, shredders, and scavengers).

of scrapers at Site 3 was linked to dominance by Heptageniidae (Ephemeroptera) and suggests an abundance of diatoms and a shortage of filamentous algae. Interestingly, as will be discussed in Sect. 6.3.3.3, Site 3 had the highest periphyton colonization rates and chlorophyll *a* standing crop in the periphyton colonization study.

Community composition varied greatly between this study and the Y-12 Plant BMAP study in three respects. First, the BMAP studies found that Chironomid relative abundance was greatest at the three sites upstream from EFK 13.8. In this study, however, Chironomids were absent at two sites in EFPC and at the reference site, and were 8% of the individuals at the remaining EFPC sites. Diptera (Chironomidae) were the most abundant insects found during the BMAP study and were the dominant taxon at five of the six EFPC sites, representing over 61% of all individuals (Hinzman 1992). Second, the BMAP studies reported that Trichopterans were rarely collected at sites above EFK 13.8 or at EFK 10.6, having relative abundances below 0.9%. Trichoptera in this study constituted 84% of individuals at Site 1, 74% at Site 2 (4.6 and 3.7 km, respectively, upstream from EFK 18.2), and 57% at Site 4 [~200 m (660 ft) upstream from EFK 10.6]. Third, Hinzman (1992) stated that mollusks were rarely collected at any EFPC site except EFK 13.8, where they were only about 2.7% of the total density. In contrast, mollusks (Gastropoda and Pelecypoda) in this study ranged from 5% to 35% of individuals at the six sites in EFPC. Reasons for these differences are unknown; however, the BMAP study has been showing a trend toward recovery of biotic resources in the upper part of the creek below Lake Reality.

The differences in community composition between the Y-12 Plant BMAP investigations and this study suggest that the benthic macroinvertebrate community composition in the sampled areas in EFPC has changed since 1987. However, this investigation supports the conclusions from the second BMAP report (Hinzman 1992) and suggests that the benthic communities in EFPC still (1) are dominated by large numbers of individuals from only a few families, (2) are dominated by tolerant organisms, (3) have less total taxa or EPT richness than the reference stream, and (4) have lower diversity than the reference stream. Thus, the benthic communities in lower EFPC appear to be environmentally stressed even though there is evidence some recovery has occurred since the late 1980s.

Abundance (density). Benthic macroinvertebrate density varied considerably among the six EFPC sites, and did not follow a consistent trend (Fig. 6.64). Site 2 had the highest density at 339/m^2, nearly twice that of the reference site (186/m^2). Sites 3, 4, and 6 yielded the lowest densities (67 to 73/m^2), whereas sites 1 and 5 had densities similar to that of the reference site. The variation in densities among the EFPC sites may be attributable to differences in physical

and/or chemical characteristics at the sites. Also, the three sites having lowest densities each had a unique feature that may have influenced benthic macroinvertebrate density. Site 3 had been physically disturbed four weeks prior to the sampling (see discussion in the "Limitations and Uncertainties" section that follows). Site 4 was downstream from the Oak Ridge Sewage Treatment Plant, and Site 6 was just downstream from the confluence of Bear Creek and EFPC.

Densities of benthic macroinvertebrates observed in this study were generally an order of magnitude less than those reported in the second BMAP study (Hinzman 1992). Several factors could account for the dramatic reduction in observed densities between the two investigations, including (1) natural seasonal variability, in combination with between-site differences in physical factors such as substrate and stream flow characteristics; (2) differences in sampling efficiencies between field crews collecting the samples; and (3) real decreases in density resulting from increased degradation of water quality.

Diversity. Family diversity, expressed as the Shannon-Wiener Diversity Index (H′), was lower at all six EFPC sites than at the reference site (Fig. 6.67). H′ was lowest at Site 1, then progressively increased downstream to Site 4 before slightly decreasing at Sites 5 and 6. The three sites in EFPC closest to the Y-12 Plant had the greatest mercury concentrations in surface water and floodplain soils, and also had the lowest diversity indices for benthic macroinvertebrates.

Evenness was greatest at the reference site (at 0.790) (Fig. 6.68). Evenness generally increased downstream from the Y-12 Plant, reaching a maximum at Site 5, then decreased slightly at Site 6 (Fig. 6.67). The low evenness indices at all the EFPC sites, especially at Sites 1 and 2, indicate that, in contrast with the reference site, benthic macroinvertebrate communities at these sites are dominated by a large proportion of individuals from only a few of the total number of families.

Diversity values and trends in this study were similar to those reported in the second BMAP study (Hinzman 1992). Both studies show that diversity (H′) was lower at every site in EFPC than at the reference site. In addition, both studies reported that the lowest diversity occurred nearest the Y-12 Plant. Lastly, both studies observed that diversity increased downstream from the Y-12 Plant, reaching a maximum at Site 4 (EFK 10.6), near the Oak Ridge Sewage Treatment Plant.

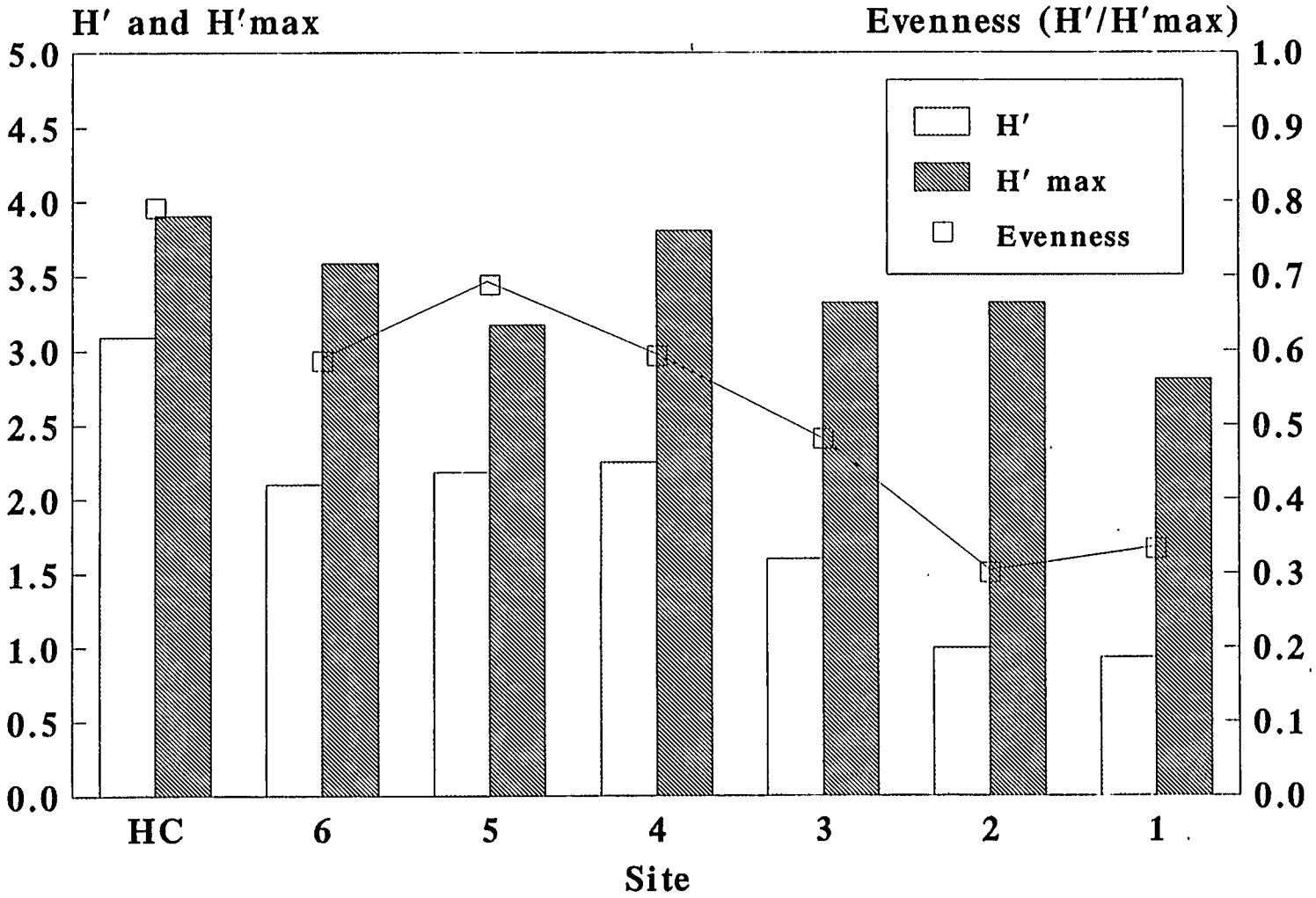

Fig. 6.67. Diversity indices and relative diversity of benthic macroinvertebrates collected from EFPC and Hinds Creek, October 22-29, 1991.

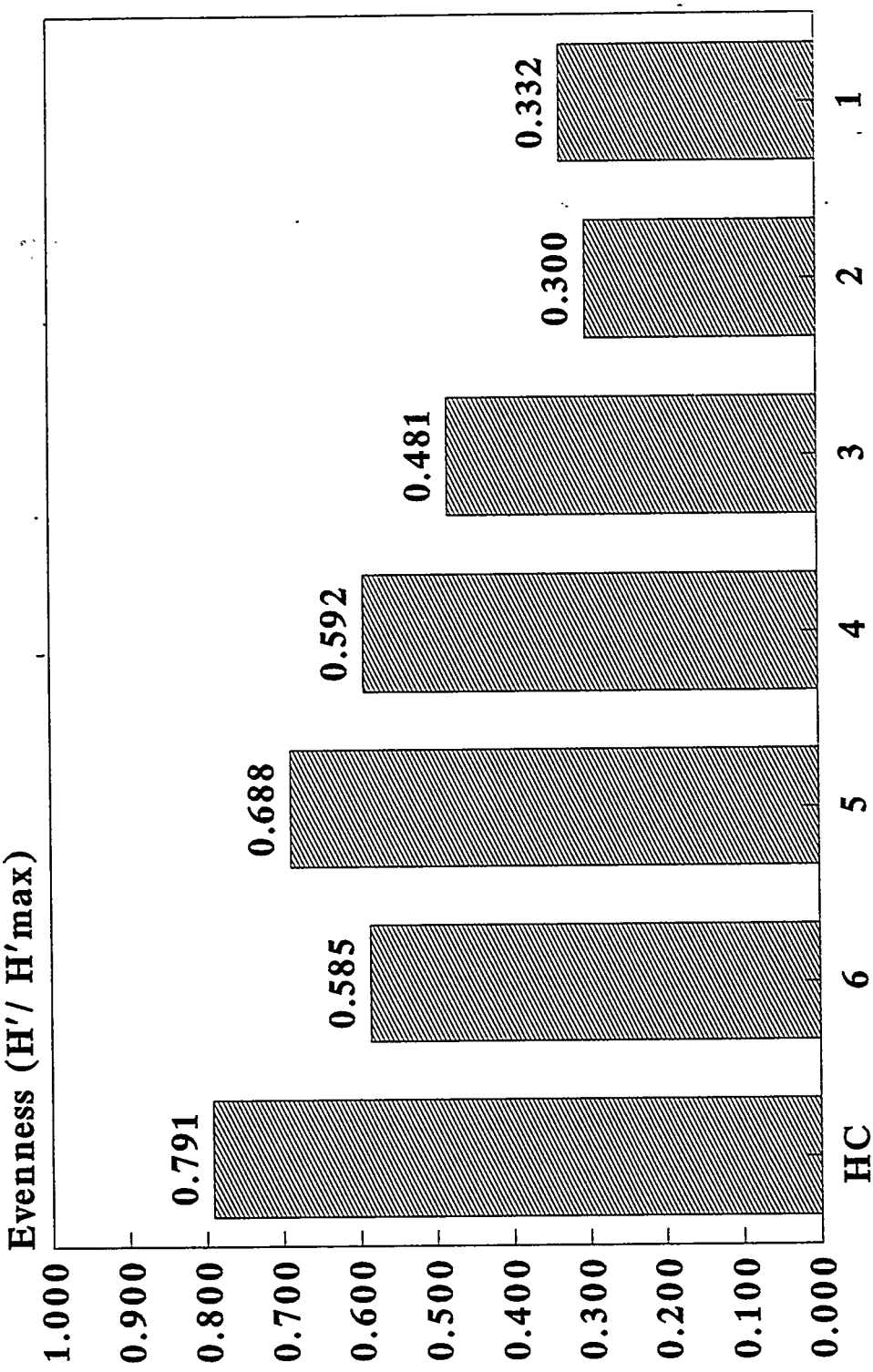

Fig. 6.68. Relative diversity of benthic macroinvertebrates collected from EFPC and Hinds Creek, October 22–29, 1991.

Limitations and uncertainties

As previously discussed in Sect. 6.3.3.1, the greatest limitation affecting interpretation of these aquatic biota sampling data is that they represent only a single point in time. A second uncertainty is the potential impacts that the electrofishing sampling (conducted approximately two weeks prior to the benthic macroinvertebrate sampling) might have had in the number and distribution of benthic macroinvertebrates. Potential impacts to benthic organisms could have occurred from direct exposure to the electrical field during sampling and from physical damage to the riffles when the instream sampling crews walked over them. However, detrimental impacts to most taxa in the benthic macroinvertebrate community are presumed to have been negligible, with the exception of impacts to aquatic crayfish, which were stunned and captured for subsequent contaminant analysis. The relatively large body size of the crayfish would make them more susceptible to the electrical field than the smaller-sized aquatic insects, crustaceans, mollusks, and Oligochaeta worms constituting the remainder of benthic macroinvertebrate taxa.

A third limitation of the data is that the actual number of Oligochaeta worms collected at each site is unknown. The sampling crews identifying the benthic organisms were not able to count the number of Oligochaeta because most of the worms were torn into segments during capture from the Surber net or collecting pans.

A fourth source of uncertainty involved the limitations of manually locating and removing organisms from the collection pans. Thoroughness of the removal of organisms was maximized by (1) working with small volumes of sediment in each collection pan, (2) using a small volume of water in each pan, (3) carefully pouring out and replacing the water if it became too turbid to see through, and (4) having a second person check over each pan before the sediment was discarded.

A final source of uncertainty specifically regards the data from Site 3. The existing riffle at this site had been physically disturbed and altered by two young boys approximately four weeks prior to the benthic sampling activity. The boys had placed numerous rocks [of ~10 to 20 cm diameter (4 to 8 in.)] and other debris across the existing riffle, creating a small dam. Impacts of these activities on the benthic macroinvertebrate community at Site 3 are not known.

6.3.3.3 Periphyton survey

The purpose of the periphyton colonization study was to obtain data on the rate of colonization and biomass increase because periphyton forms part of the base or first trophic level

of the food chain in stream habitats. Periphyton is the primary food for stonerollers, one of the fish indicator species in this ERA. Its reestablishment after disturbance may be a critical factor in the recovery rates of species populations at higher trophic levels (Steinman and McIntire 1990).

Sampling methods

Periphyton biomass and colonization rates were measured in riffle habitats at six sites in EFPC and Hinds Creek during October 31 through December 12, 1991, using artificial substrates according to technical procedure *TP-309-6: Periphyton Sampling Using Artificial Substrates* (LWA 1990). The riffle locations were all within the stream segments where fish populations and benthic macroinvertebrates were sampled. Chlorophyll *a* and ash-free dry weight were measured to assess the algal and total periphyton components, respectively. Unglazed ceramic tiles (5.8 cm^2) were attached to building bricks with silicone glue, then secured in the riffles. Triplicate sets of tiles were removed from each brick for chlorophyll *a* extraction and for ash-free dry weight determination after 14, 21, and 37 d of exposure at most sites. However, exposure times at Site 1 were 18, 25, and 41 d. Also, the tiles at Site 3 and at Hinds Creek were lost during rainstorms; therefore, no data were collected for the 37-d colonization for these two sites.

Colonization rate (μg chlorophyll $a/cm^2/d$) was calculated by dividing the μg chlorophyll *a* per cm^2 of tile surface area by the number of days prior to removal (i.e., 14 or 18 d).

Several physical and chemical field measurements were collected weekly. Field measurements included temperature, pH, conductivity, dissolved oxygen, and flow rates and stream depths across the riffle. In addition, a 24-h integrated measurement of light intensity was obtained in the riffles within 30 cm (12 in.) of the position where the artificial substrates were placed.

Results

Environmental conditions. Stream physical and morphological characteristics at the riffle locations during the periphyton colonization study are summarized in Table 6.55. Mean values of pH, conductivity, and sunlight intensity were fairly uniform across all six sample sites in EFPC and at the reference site. Mean water temperatures were greater at sites in EFPC closest to the Y-12 Plant; temperatures at Sites 5 and 6 were significantly less than at Site 1 (t-test, $P < 0.05$). In addition, mean water temperature at the reference site was significantly lower than mean water temperatures at all sampling sites in EFPC except Sites 6 and 3. Mean stream flows at actual brick locations at Sites 1, 4, and 5 ranged from 2.4 to 2.9 ft/s; values ranged from 1.3

Table 6.55. Summary of stream physical and chemical characteristics in the riffles in EFPC and Hinds Creek where periphyton colonization study was performed, October 31–December 13, 1991[a]

Parameter	Sampling location						
	1	2	3	4	5	6	HC
Temperature (°C)	16.4 ± 4.3 (12.5-22.9) 5	14.0 ± 2.5 (11.6-15.0) 5	10.8 ± 3.2 (8.7-15.5) 4	12.3 ± 2.1 (10.2-15.6) 5	11.5±2.1 (9.9-15.0) 5	9.7 ± 2.7 (7.0-13.7) 5	7.4 ± 2.0 (5.0-11.1) 4
pH	8.01 (7.78-8.56) 5	8.04 (7.85-8.51) 5	8.00 (7.7-8.47) 4	7.58 (7.72-8.15) 5	7.52 (7.12-8.33) 4	7.99 (7.8-8.5) 4	8.08 (7.9-8.36) 4
Dissolved oxygen (DO) (mg/L)	9.4 ± 0.8 (8.8-10.6) 5	9.9 ± 0.8 (8.8-10.8) 5	11.5 ± 2.0 (9.0-13.2) 4	10.3 ± 1.1 (8.8-11.4) 5	11.1 ± 1.2 (10.0-13.2) 5	11.0 ± 1.3 (10.2-13.4) 5	12.3 ± 1.0 (10.6-13.0) 4
Conductivity (μmhos/cm^2)	466 ± 17 (440-480) 5	508 ± 65 (440-600) 5	505 ± 73 (450-610) 4	504 ± 91 (360-600) 5	432 ± 58 (330-470) 5	424 ± 88 (280-520) 5	385 ± 7.0 (300-47.0) 4
Mid-stream flow (ft/sec)	2.9 ± 1.3 (1.6-5.1) 5	1.3 ± 0.7 (0.5-2.1) 5	1.4 ± 0.4 (0.8-1.8) 3	2.5 ± 1.6 (1.0-5.3) 5	2.4 ± 0.9 (1.6-3.7) 4	1.6 ± 0.6 (0.8-2.1) 4	1.4 ± 0.0 (1.0-1.8) 3
Mid-stream depth (m)	0.15 ± 0.00 (all were 0.15) 5	0.19 ± 0.02 (0.15-0.21) 5	0.20 ± 0.03 (0.18-0.24) 4	0.22 ± 0.08 (0.12-0.30) 5	0.20 ± 0.08 (0.12-0.30) 4	0.27 ± 0.13 (0.09-0.38) 5	0.11 ± 0.0 (0.09-0.01) 4
Stream width (m)	6.1 ± 0.5 (5.5-6.4) 5	5.9 ± 1.0 (5.2-7.6) 5	4.5 ± 0.3 (4.3-4.9) 4	5.3 ± 1.5 (4.3-7.9) 5	9.6 ± 1.5 (8.2-11.4) 4	(16.5 ± 0.9 (15.2-17.7) 5	6.4 ± 0.0 (6.1-7.0) 4
Light intensity (E/m^2)	7.2 ± 1.0 2	7.6 ± 0.4 2	6.5 1	7.9 ± 0.0 2	7.9 ± 0.0 2	NC[b]	7.4 1

[a]Values are mean ± standard deviation, range in parenthesis, and sample size.
[b]NC = Measurements not obtained due to rainstorm ruining the ozalid meters.

to 1.6 ft/s among the remaining sites. At the time of the study, canopy cover was minimal at all sites because the tree leaves had fallen.

Colonization rate. Periphyton colonization rate varied considerably among the sites in EFPC, ranging from 0.006 to 0.712 μg chlorophyll *a* per cm^2 per d (Fig. 6.69). Four of the sites in EFPC, including the three sites closest to the Y-12 Plant, had colonization rates greater than or equal to the rate at the reference site. Site 3 had the highest colonization rate, more than sevenfold greater than at the reference site. The lowest colonization rate was observed at Site 4. The site furthest downstream from Y-12, Site 6, had a colonization rate ~50% greater than that of the reference site.

Periphyton colonization rate is influenced by many factors, including concentrations of dissolved nutrients, current velocity, irradiance, substrate, grazing pressure, and species composition of the propagule pool (the physical source of individuals potentially available to colonize a new substrate). The propagule pool directly relates to the species availability, dispersal ability, and performance regarding ecophysiology, life history strategies, and competitive abilities, and is also influenced by instream physical and chemical conditions. In this study, the influence of several of these factors should have been fairly uniform among the sites in EFPC. For example, identical substrate material and configurations were used at all sites. Mean measurements for depth and flow rate at each substrate location were also fairly uniform among the sites. Light intensities at the substrate locations were also similar, yet show an inverse rank correlation with periphyton colonization rate. Although mean water temperatures at the reference site were lower than at several sites in EFPC, colonization rates and mean water temperatures did not display consistent trends. Thus, it is likely that factors other than water temperature were responsible for the observed results.

The high colonization rate at Sites 1, 3, and 6 suggests that the propagule pool is dominated by pioneer species having high reproduction rates and short generation times, whereas Sites 4 and 5 are populated by more effective late colonist species.

Standing Crop. Periphyton standing stock, or biomass, growth in EFPC, whether expressed as ash-free dry weight (AFDW) or chlorophyll *a*/cm^2, varied among sites in EFPC (Fig. 6.70) and generally followed the same pattern observed for colonization rates. For both measures, only Sites 4 and 5 in EFPC were lower after 14 d than at the reference site. However, after 21 d, both measures at Sites 3, 4, 5, and 6 were much lower than at the reference site, with the exception that AFDW at Site 4 did exceed the reference site.

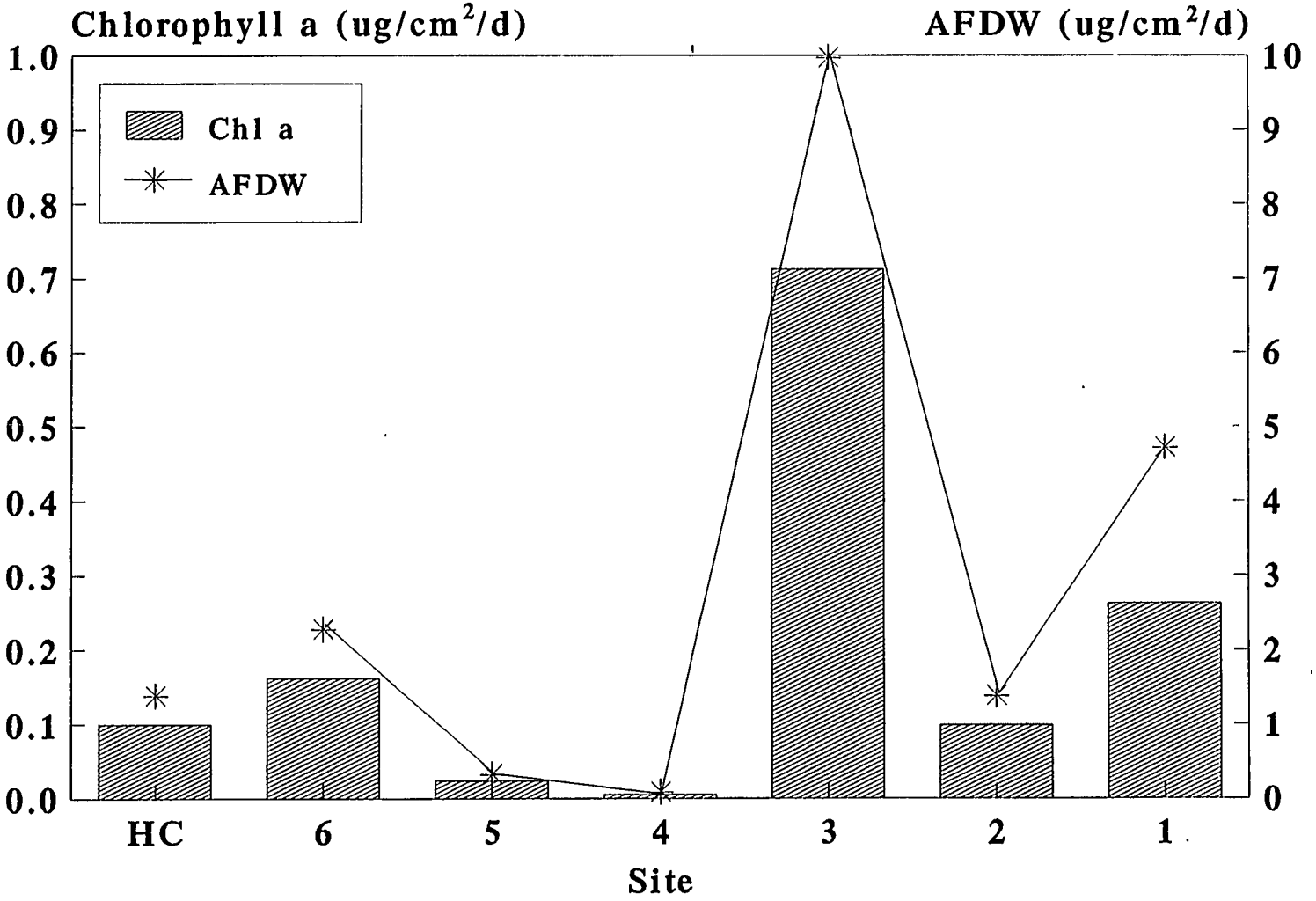

Fig. 6.69. Periphyton colonization rates [expressed as μg chlorophyll *a* per cm² per d and Ash-Free dry weight (AFDW) per cm² per d] for sites in EFPC and Hinds Creek.

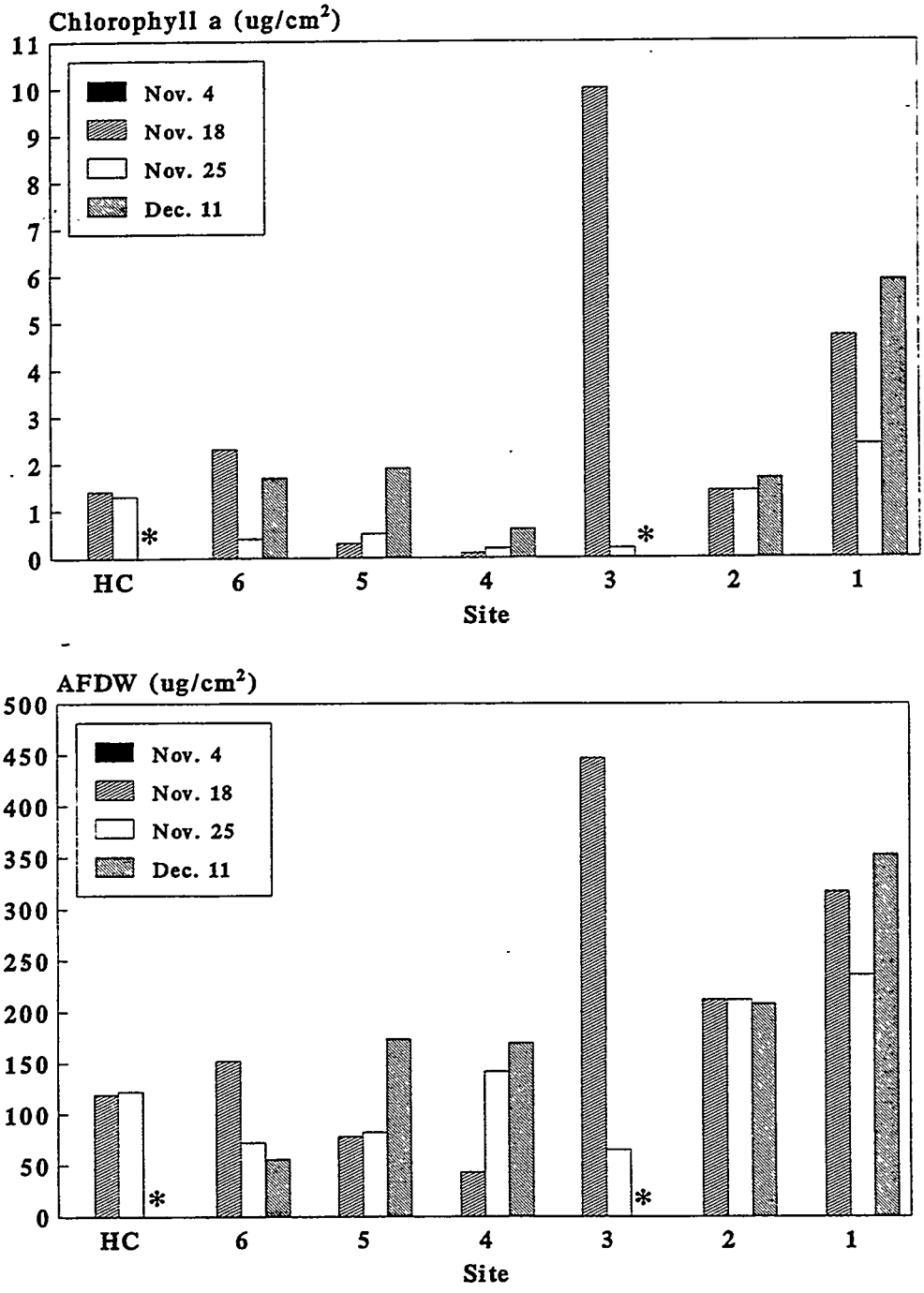

Fig. 6.70. Periphyton growth in EFPC and Hinds Creek, expressed as μg chlorophyll *a* per cm^2 and as μg ash-free dry weight (AFDW) per cm^2.

92-164P/041793/ERA

In general, periphyton biomass was greater at the three sites closest to the Y-12 Plant (Fig. 6.70). This trend was also reported in the second BMAP report for the Y-12 Plant after examination of periphyton attached to rocks in EFPC (Hinzman 1992). The BMAP also observed that AFDW was variable among the sites in EFPC, but lowest at EFK 10.6 (very near Site 4). A nine-week periphyton colonization study of was conducted at four of the BMAP sampling sites during June through August 1988 (Hinzman 1992). BMAP reported nearly 10 μg chlorophyll *a*/cm^2 periphyton colonization after five weeks at EFK 23.2 (upstream from Site 1), an amount similar to that observed in this study (5.9 μg chlorophyll *a*/cm^2). Further direct comparison of chlorophyll *a* or AFDW measurements in this study with results from the BMAP investigations is restricted because of the short duration of this colonization study (due to loss of tiles from major storm event) versus the long-term growth reported in the BMAP results.

Although the greater growth of periphyton at sites closest to the Y-12 Plant might suggest that these sites have unstressed, structurally and functionally desirable periphyton communities, the opposite may actually be true. Periphyton communities in highly stressed environments appear to be more resistant to disturbance than those growing in low-stress environments (Steinman and McIntire 1990) and are quicker to recover, in terms of biomass. However, it is unknown whether periphyton taxonomic structure and functional processes recover as quickly. Therefore, periphyton biomass may not be the best indicator of lotic ecosystem recovery.

Limitations and uncertainties

The greatest limitation of this study is that its duration was only five weeks, which is a fairly short period for assessing colonization and growth on new, bare substrates. The study was originally scheduled to last several weeks longer, but it was interrupted by major rainstorms between November 30 through December 3, 1991, that washed away all of the substrate tiles at the reference site and at Site 3 in EFPC.

A second source of uncertainty affecting both the chlorophyll *a* and AFDW data is the possible loss of material from the tiles during (1) removal of the tiles from the bricks by field personnel, (2) transport of the tiles from the field to the laboratory, and (3) handling of the tiles by personnel conducting the analyses in the laboratory. During removal of the tiles from the bricks, some periphyton was occasionally scraped off the tiles when part of the latex gloves worn by the field personnel accidentally brushed against the edge of the tiles.

6.3.3.4 Two-component aquatic relationships

"Two-component relationships" refers to how one group behaves in the presence of another (e.g., fish and benthic macroinvertebrates). This analysis is the beginning of weight-of-evidence analysis, which is fully developed in Sect. 6.4.

Fish and benthic macroinvertebrates

The numbers of benthic macroinvertebrate families and fish species followed similar trends among the sites in EFPC as well as in relation to the reference site (Fig. 6.71). Total richness for both generally increased downstream from the Y-12 Plant, but was still lower at all EFPC sites than at the reference site. Although the trend toward increasing numbers of taxa downstream from the Y-12 Plant suggests an improvement in water quality or habitat characteristics downstream from the plant, the depressed number of benthic macroinvertebrate families and fish species at all EFPC sites relative to the reference site reinforces the conclusion that, overall, EFPC is negatively affected.

Benthic macroinvertebrate EPT richness and the percentage of fish species classified as intolerant (i.e., sensitive to degraded environmental conditions) generally followed similar trends among the sites in EFPC, as well as in relation to the reference site (Fig. 6.72). The percentages of intolerant fish species and EPT richness were lowest at the site nearest the Y-12 Plant. The percentage of intolerant fish species steadily increased downstream, with percentages at Sites 5 and 6 slightly greater than at the reference site. EPT richness at all EFPC sites downstream from Site 1 was three or fourfold greater than at Site 1; however, EPT richness at all sites in EFPC was less than at the reference site. The low number of benthic macroinvertebrate EPT families and intolerant fish species in EFPC relative to the reference stream indicates that EFPC is negatively affected by degraded water quality and/or habitat conditions.

Benthic macroinvertebrate and fish diversity indices (H′) followed similar trends among the sites in EFPC (Fig. 6.73), but differed slightly in relation to the reference site. For both taxa groups, H′ was lowest at the three sites nearest the Y-12 Plant, but generally increased downstream. Although benthic macroinvertebrate H′ was less at all sites in EFPC than at the reference site, fish H′ at Site 6 was slightly greater than at the reference site. The clear trend of increasing diversity indices with distance downstream from the Y-12 Plant for two major groups of indicator organisms, coupled with lower indices for all sites in EFPC than at the reference site (except at Site 6 for fish), suggests that EFPC is negatively affected.

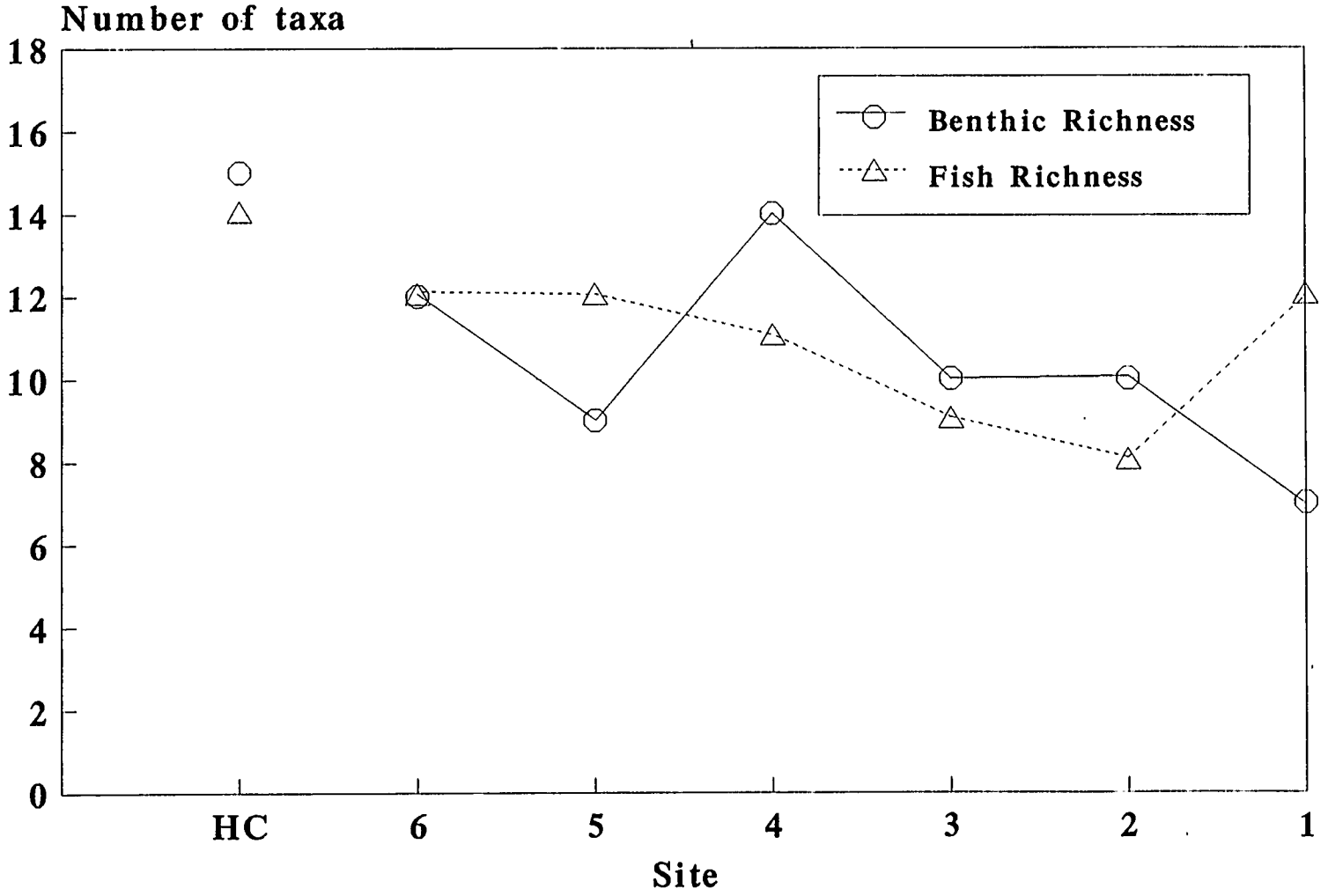

Fig. 6.71. Number of benthic macroinvertebrate families and fish species collected at each sample site in EFPC and Hinds Creek, October 7-29, 1991.

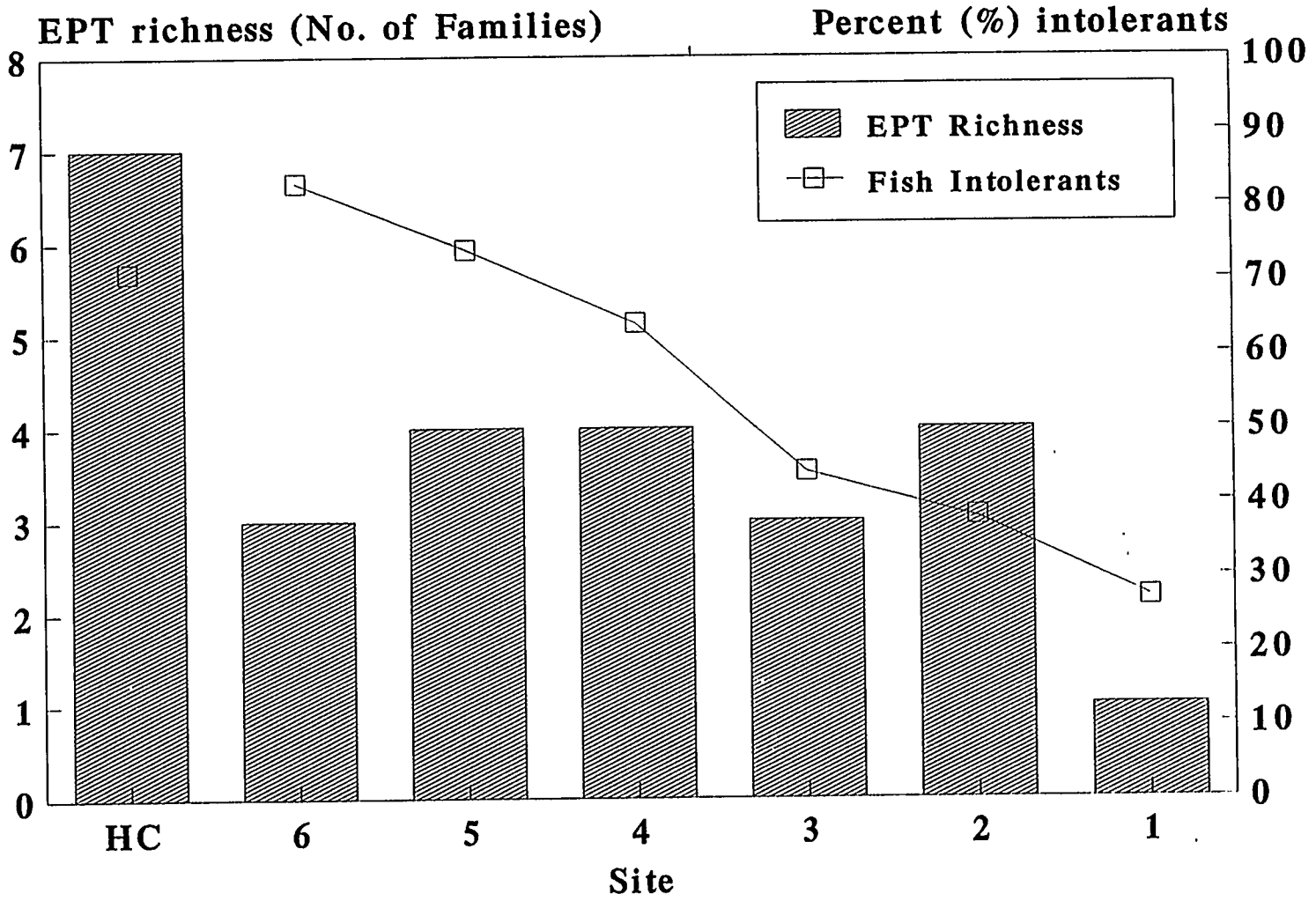

Fig. 6.72. Benthic macroinvertebrate EPT richness and percentage of intolerant fish species collected at each site in EFPC and Hinds Creek, October 7–29, 1991.

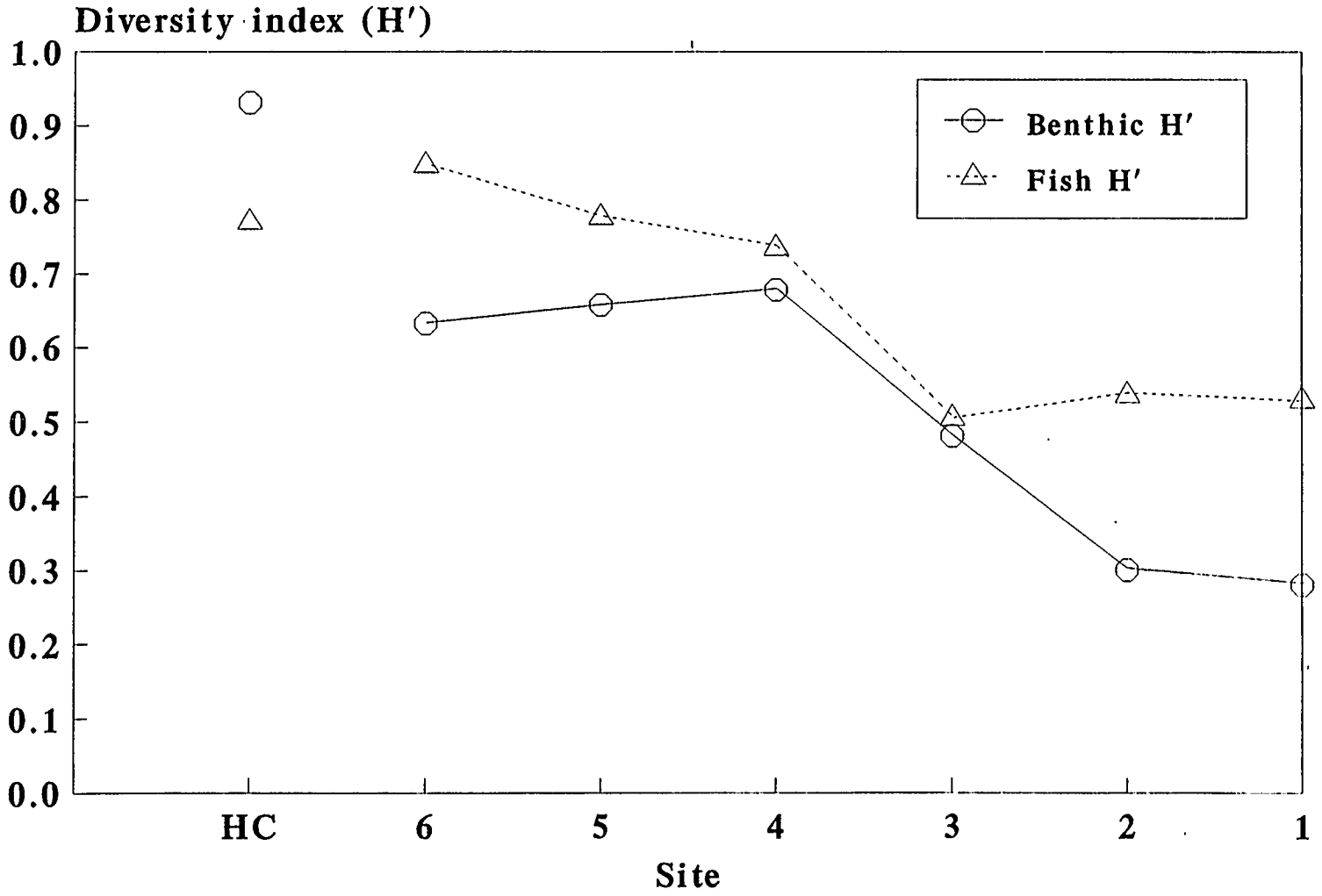

Fig. 6.73. Shannon-Wiener diversity indices for benthic macroinvertebrates and fish collected at sites in EFPC and Hinds Creek, October 7–29, 1991.

Fish or benthic macroinvertebrates and periphyton

No clear trends were observed regarding periphyton colonization rate or biomass and (1) relative abundance or density of benthic scrapers and grazers or (2) abundance of stonerollers. The lack of clear relationship could be an artifact of the study design. The periphyton colonization study was biased for measurement of species favoring early colonization of bare surfaces, which may not necessarily be the preferred food source for the stonerollers and the benthic grazers and scrapers.

6.3.3.5 Small mammal survey

Survey methods

Six sites along EFPC were selected for population surveys of small mammals (Figs. 6.3 through 6.8). A seventh site at Mill Branch within the EFPC watershed was selected as a reference site (Fig. 6.10). A small mammal trapping grid was established within each of the larger bird-survey units. Trapping grids consisted of 24 trap stations arranged in either a 6 × 4 or a 3 × 8 configuration, depending upon floodplain width and physical considerations. Spacing between trap stations was 10 m (33 ft); a single trap was placed at each station. Grids were situated so as to maintain each within a single habitat type while retaining a right-angle orientation to the stream channel. Trapping grids were limited to one side of the stream at the reference site and at Sites 3, 4, and 6.

Sherman single-capture live traps, having dimensions of 8 × 8 × 25 cm, were used. Bait was a standard mixture of oats and peanut butter. Bits of sardines were added during the third trapping session to attract short-tailed shrews (*Blarina brevicauda*) (after Talmage and Walton 1990). The survey period ran from October 15, 1991, through November 1, 1991. Trapping sessions were operated on a four-d-open, three-d-closed cycle. Captured animals were identified according to species, sex, and age by pelage, when possible, weighed, toe-clipped, and released. Recaptures were identified by unique toe-clip.

An abundance index (CE_o) based on catch/effort was calculated for each site. Effort was measured in trap night units (TU). This index, corrected for sprung traps after Nelson and Clark (1973), allows direct comparison of abundances among sites.

Table 6.56 presents these values along with numbers of original captures (C_o) and total captures (C_{TL}). Trap activity is indicated by total catch/effort (CE_{TL}). Unless noted, calculated values are for all species; captures of species other than the white-footed mouse (*Peromyscus leucopus*) were rare.

Results

Four small mammal species were captured among the seven sites. These included the white-footed mouse, short-tailed shrew, pine vole, and eastern chipmunk (*Tamias striatus*). Not all species were captured at each site. Captures were skewed heavily toward the white-footed mouse, with only Site 6 lacking a white-footed mouse capture. The short-tailed shrew was captured only at Sites 4 and 5. Pine vole and eastern chipmunk were captured only at Site 4.

Sites 1, 4, and 5 exhibited very similar values for the abundance of all small mammal species (Table 6.57). Although Site 1 had the highest abundance index (at 5.25), only the white-footed mouse was captured at this site. Differences in habitat diversity, the length of edge or boundary between habitat types and short interhabitat patch distances could account for the relatively high abundance index generated at Site 1. Site 4, having an abundance index of 3.44, had a total of four captured species. The species with the greatest number of individuals captured was the white-footed mouse. Site 5 had captures of the white-footed mouse and short-tailed shrew, and an abundance index of 4.19.

Sites 2 and 3 yielded abundance indices of 1.88 and 1.97, respectively. Both had captures of only the white-footed mouse. Although Site 2 yielded only four individuals, the third lowest number of white-footed mice captured, the abundance index was fourth highest of the seven sites. An unusually high percentage of traps were disturbed throughout the trapping period at this site, often approaching 80% on any given night. The habitat at Site 3 was somewhat different from the other sites—an early-stage old field. Areas in a slightly later successional stage located in an adjacent county yielded abundance indices of 3.49 and 3.41 during February 1991, and included the golden mouse (*Peromyscus nutalli*) and the harvest mouse (*Reithrodontomys humulis*) (Hnilicka and Wenzel 1991). At Site 3, several traps were visited but left untripped. The fecal pellets on and within these traps indicated that these visits probably were made by harvest mice. The habitat at this site contained a large percentage of grass cover that could be expected to be inhabited by pine voles. The absence of this species, as well as that of the short-tailed shrew,

Table 6.56. Catch per effort, species richness, species diversity, and species evenness of small mammals along EFPC

Parameter		Sampling site						
		1	2	3	4	5	6	Mill Branch
CE_O[a]	x	5.25	1.88	1.91	3.44	4.19	0.37	1.11
	s	5.80	2.72	2.26	2.00	3.43	1.23	1.93
CE_{TL}[b]	x	21.57	13.82	3.78	4.98	15.57	0.37	2.96
	s	9.79	5.15	4.48	3.08	5.78	1.23	2.77
SR[c]		1	1	1	4	2	1	1
H'[d]		0.00	0.00	0.00	1.37	0.66	0.00	0.00
J[e]		0.00	0.00	0.00	0.99	0.95	0.00	0.00

[a]CE_O = catch/effort of original captures.
[b]CE_{TL} = catch/effort of total captures.
[c]SR = species richness.
[d]H' = species diversity.
[e]J = species evenness.

CE = A x 100 ÷ (TU - IS/2)
where
CE = catch/effort
A = no. of animals
TU = P x I x N
P = no. of trapping intervals
I = length of trapping interval
N = no. of traps
S = total traps tripped

x = mean
s = standard deviation

Table 6.57. Abundance index of small mammals along EFPC based on calculations of catch per effort

Parameter	Sampling site						
	1	2	3	4	5	6	Mill Branch
C_O[a]	13	4	5	9	11	1	3
C_{TL}[b]	54	25	10	13	40	1	8
Effort[c]	254.5	190	274	267	259	264	276
CE_O[d]	5.25	1.88	1.91	3.44	4.19	0.37	1.11
CE_{TL}[e]	21.57	13.82	3.78	4.98	15.57	0.37	2.96

[a]C_O = original captures.
[b]C_{TL} = total captures (original + recapture).
[c]Effort [(Trapping Units (TU)] = trap nights. Effort corrected for sprung traps after Nelson and Clark (1973). Corrections were made for tripped traps and traps rendered inoperative because of disturbance (overturned, turned on side, turned on end, or stolen). Rarely, a trap would be intentionally left inoperative because of a birthing female using the trap as a nest or because of an excessively trap-prone individual reentering a trap immediately upon release.
[d]CE_O = catch/effort of original captures.
[e]CE_{TL} = catch/effort of total captures.

$CE = A \times 100 \div (TU - IS/2)$
where
CE = catch effort
A = no. of animals
TU = P x I x N
P = no. of trapping intervals
I = length of trapping interval
N = no. of traps
S = total traps tripped

is notable. A previous study involving the trapping and removal of short-tailed shrews from a portion of this site was conducted a year prior to this survey.

A single short-tailed shrew was captured once at Site 6. The resulting abundance index (0.37) was the lowest exhibited in the survey. The absence of other species of small mammals at this site, especially of the white-footed mouse, is notable. In a similar study performed in an adjacent county during February 1991, Hnilicka and Wenzel (1991) reported abundance indices of 2.86 and 0.60 from grids located in a three-year-old sweetgum plantation where the groundcover had been controlled by herbicide application.

The reference site at Mill Branch yielded an abundance index of 1.11, roughly three times that of Site 6 but only one-fourth of that of Site 1. Only the white-footed mouse was present at the Mill Branch site.

Contaminant concentrations

Results are summarized for average concentrations of arsenic, chromium, mercury, and zinc in the soil at-each of the six survey sites along EFPC. Arsenic shows a nearly constant level along the course of the stream, with only a slight elevation at Site 3, an area of suspected high deposition. Concentrations of the other contaminants (aluminum, mercury, and zinc) exhibit a general trend of reduction in a downstream gradient. A mercury concentration spike occurs at Site 3, whereas zinc is elevated at Site 4 and is found at decreased but still-elevated levels through Site 6. Cumulatively, these contaminants also show a downstream concentration gradient, with the exception of Site 3. Small mammal relative abundance indices show an inverse relationship with cumulative contaminant concentrations at Sites 2 through 4. Site 1 showed a high relative abundance index along with an especially high average concentration of mercury in the soil. Site 6 generated a low relative abundance index, although concentrations of these four contaminants are lowest at this site.

Conclusions

The distribution of small mammal species and individuals captured within the sampling grids along EFPC exhibit no consistent relationship with soil contaminant concentrations. The low species richness and the high population of small mammals encountered at Site 1 and the site's high mercury concentrations could suggest a stressed system. Species richness was consistently low among the sites, with only two sites exhibiting more than one species. The absence of the short-tailed shrew from five of the sites is notable. Although some researchers have concluded that distributions of certain species of shrews are limited by hydric conditions

because of their fossorial nature, the soil conditions at most sites would not be expected to exclude shrews. Site 3 was shown to have supported short-tailed shrews during a prior unrelated study. Also of note is the absence of voles at Site 3. The meadowlike conditions throughout much of the grid could be expected to support a substantial population of voles. A 1985 survey of a similar old-field area along the Clinch River in adjacent Roane County showed a healthy population of voles. Populations of small mammals, especially voles, exhibit cyclic increases and decreases in abundance. The local vole population could have been at a low point during the survey period. The subjective consensus of other researchers in the southeast was that small mammal populations as a whole were generally depressed during 1991 (Chason 1991).

The vegetation structure and composition at Mill Branch, being more upland in nature, was atypical of many of the sites along EFPC. However, this difference was not expected to significantly affect the survey, because the target species are fairly ubiquitous. Differences were expected to appear in the relative abundance indices, although not in the direction exhibited.

6.3.3.6 Bird survey

Bird populations on the lower EFPC floodplain were studied in the fall season. During this time of year in Tennessee, most birds are not spatially distributed in territories. They wander and often group in flocks. On any one day there may be no individuals of a species present on a given site, whereas on the next, a flock of several individuals may be present. It was not feasible to take a sufficient number of samples to reduce the high variance in the population numbers on the sites. The data do illustrate, however, the variety of species encountered, especially migrants, and indicate possible trends in abundance.

Survey methods

Birds were counted at six sites on EFPC (Figs. 6.3 through 6.8) and at one reference site on Mill Branch (Fig. 6.10). The sample plot method was used for sampling at five of the EFPC sites (Sites 2, 3, 4, 5, and 6) and at the reference site because the floodplains were large enough for a 1 ha (2.47 acres) sample plot. Site 1 was sampled by the belt transect method because the floodplain was too narrow [23 m (75 ft)] at this location. The size of the belt transect at Site 1 was 23 m (75 ft) × 46 m (151 ft).

Eleven counts were made at all sites between September 26 and November 10, 1991. The count period began at sunrise and extended until approximately 10:00 a.m. All birds identified by sight or sound during the count were recorded.

Results

Table 6.58 lists the 552 birds from 45 species that were counted on Sites 2, 3, 4, 5, and 6 on EFPC and on the Mill Branch reference site. Because of the low numbers of individuals of some species and the fact that many species were transient, only those species that are known to be permanent residents and that were found in relatively high numbers (i.e., >25 birds counted during the study) were evaluated for possible trends. These species, hereafter designated as "high-population species," are listed in Table 6.59.

As shown in Table 6.59, Sites 2, 3, 4, 5, and 6 on EFPC had higher total numbers of individuals of high-population species than did the Mill Branch reference site, with Sites 2 and 3 having over twice as many birds, and Site 4 having over three times as many. This is probably because the Mill Branch site had a predominantly upland mesophytic plant community habitat rather than the lowland bottomland habitat found along EFPC. Lowland bottomland habitats typically have higher bird populations than upland areas. As noted earlier, the lack of a suitable reference area for the terrestrial biotic study hindered comparisons of faunal composition, but not of chemical body burden with EFPC.

Comparing bird population data with contaminant concentrations along EFPC reveals that bird populations tend to be higher in the more contaminated areas. However, these differences are probably attributable to the diversity of habitats in these areas. In areas where diversity of habitat was highest, the numbers of bird species and individual birds were also highest. Table 6.59 shows that total numbers of individuals for the two species with the largest populations—the Carolina chickadee (*Parus carolinensis*) and Carolina wren (*Thyrothorus ludovicianus*)—were higher on Sites 2, 3, and 4 (the most contaminated sites at which birds were counted) than on Sites 5 and 6 (the least contaminated EFPC sites) and at the Mill Branch reference site. An exception to this trend was the Carolina wren, whose numbers were slightly higher on Site 6 than on Sites 2, 3, and 4.

Thirty-nine individuals and ten species were counted on Site 1 (Table 6.60). Because the birds on Site 1 were counted by a different sampling method using a different sample size area than that for other sites, the results cannot be compared directly with data from the other sites. However, the data do indicate that birds were using the area and that some of the most abundant

Table 6.58. Total number of birds counted by sample plot method at sites 2 through 6 at EFPC and Mill Branch

Species	Sampling location						Total
	Site 2	Site 3	Site 4	Site 5	Site 6	Mill Branch	
Wood Duck *(Aix sponsa)*	0	0	0	0	1	0	1
Red-shouldered Hawk *(Buteo lineatus)*	1	0	0	0	0	0	1
Broad-winged Hawk *(Buteo platypterus)*	0	0	0	0	0	1	1
Killdeer *(Charadrius vociferus)*	1	0	0	0	0	0	1
Mourning Dove *(Zenaida macroura)*	0	0	0	0	0	2	2
Yellow-billed Cuckoo *(Coccyzus americanus)*	0	1	0	1	1	1	4
Chimney Swift *(Chaetura pelagica)*	0	0	1	1	0	0	2
Belted Kingfisher *(Ceryle alcyon)*	0	0	0	0	3	1	4
Red-bellied Woodpecker *(Melanerpes carolinus)*	9	0	7	0	3	7	26
Yellow-bellied Sapsucker *(Sphyrapicus varius)*	1	0	0	0	0	0	1
Downy Woodpecker *(Picoides pubescens)*	7	0	7	5	5	6	30
Hairy Woodpecker *(Picoides villosus)*	0	0	2	0	1	2	5
Northern Flicker *(Colaptes auratus)*	4	1	2	0	0	3	10
Pileated Woodpecker *(Drocopus pileatus)*	0	1	1	0	0	1	3
Eastern Wood Pewee *(Contopus virens)*	0	0	1	1	0	0	2
Eastern Phoebe *(Sayornis pheoebe)*	0	2	0	0	0	0	2
Blue Jay *(Cyanocitta cristata)*	16	23	6	6	4	5	60
American Crow *(Corvus branchyrhynchos)*	2	8	3	0	3	5	21
Carolina Chickadee *(Parus Carolinesis)*	15	20	26	8	5	10	84
Tufted Titmouse *(Parus bicolor)*	9	0	12	7	2	1	31
White-breasted Nuthatch *(Sitta carolinensis)*	0	0	2	0	0	1	3

Table 6.58. (continued)

Species	Sampling location						Total
	Site 2	Site 3	Site 4	Site 5	Site 6	Mill Branch	
Carolina Wren *(Thryothorus ludovicianus)*	20	20	23	13	26	5	107
Ruby-crowned Kinglet *(Regulus calendula)*	0	0	0	0	1	0	1
American Robin *(Turdus migratorius)*	4	5	3	0	0	0	12
Northern Mockingbird *(Mimus polyglottos)*	0	0	1	0	0	0	1
Brown Thrasher *(Toxostoma rufum)*	0	0	0	1	4	0	5
European Starling *(Sturnus vulgaris)*	0	0	1	0	0	0	1
Tennessee Warbler *(Vermivora peregrina)*	0	1	0	0	0	0	1
Chestnut-sided Warbler *(Dendroica pensylvanica)*	1	0	0	0	0	0	1
Magnolia Warbler *(Dendroica magnolia)*	1	4	0	0	0	2	7
Yellow-rumped Warbler *(Dendroica coronata)*	0	0	0	1	2	0	3
Yellow-throated Warbler *(Dendroica dominica)*	0	1	0	0	0	0	1
Pine Warbler *(Dendroica pinus)*	2	0	0	6	0	1	9
Palm Warbler *(Dendroica palmarum)*	0	1	0	0	0	0	1
Bay-breasted Warbler *(Dendroica castanea)*	0	1	2	0	0	8	11
Black and White Warbler *(Mniotilta varia)*	0	0	1	0	0	1	2
American Redstart *(Setophaga ruticilla)*	0	1	0	0	0	0	1
Ovenbird *(Seirus acurocapillus)*	0	2	1	0	0	0	3
Common Yellowthroat *(Geothlypis trichas)*	0	2	0	0	0	0	2
Hooded Warbler *(Wilsonia citrina)*	0	0	0	0	1	0	1
Canada Warbler *(Wilsonia canadensis)*	0	0	0	1	0	0	1
Northern Cardinal *(Cardinalis cardinalis)*	9	7	25	4	13	0	58

Table 6.58. (continued)

Species	Sampling location						Total
	Site 2	Site 3	Site 4	Site 5	Site 6	Mill Branch	
Rose-breasted Grosbeak *(Pheucticus ludovicianus)*	3	6	0	2	2	4	17
Rufus-sided Towhee *(Pipilo erythrophthalmus)*	0	0	5	0	1	0	6
Red-winged Blackbird *(Agelaius phoeniceus)*	0	6	0	0	0	0	6
TOTAL	105	113	132	57	78	67	552

Table 6.59. Total number of birds counted by sample plot method at sites 2 through 6 at EFPC and Mill Branch, whose sum total for each site was greater than 25 birds

Species	Sampling location						Total
	Site 2	Site 3	Site 4	Site 5	Site 6	Mill Branch	
Red-bellied Woodpecker	9	0	7	0	3	7	26
Downy Woodpecker	7	0	7	5	5	6	30
Blue Jay	16	23	6	6	4	5	60
Carolina Chickadee	15	20	26	8	5	10	84
Tufted Titmouse	9	0	12	7	2	1	31
Carolina Wren	20	20	23	13	26	5	107
Northern Cardinal	9	7	25	4	13	0	58
TOTAL	85	70	106	43	58	34	396

Table 6.60. Total number of birds counted by belt transect method at Site 1 at EFPC

Species	Total
Belted Kingfisher	1
Downy Woodpecker	5
Hairy Woodpecker	1
Eastern Phoebe	1
Blue Jay	1
Carolina Chickadee	9
Tufted Titmouse	6
Carolina Wren	9
Northern Cardinal	5
Rose-breasted Grosbeak	1
TOTAL	39

species of birds on other EFPC sites (i.e., the Carolina chickadee, tufted titmouse, and Carolina wren) were also the most abundant birds on Site 1.

Throughout the year, ~260 species of birds could be found in the EFPC floodplain (Howell and Monroe 1957). Although bird surveys were not conducted during the spring, summer, and winter months, it is important to consider the potential problems birds face during these periods. In the late spring, summer, and early fall, birds along EFPC whose diet depends primarily on small invertebrates (i.e., primarily insects) would receive the highest dose of contaminants, because their food source is contaminated, and bioaccumulation would cause contaminant concentrations to increase in bird tissue over time. During the spring and summer months, when many birds are nesting and raising young, insect abundance is also greatest and, during this critical period in the bird's lives, insects are their primary food source. In late fall, winter, and early spring, many of the same bird species and others, which are only winter residents, would feed on seeds and other plant tissue. These birds would receive lower contaminant doses at this time of year since plant tissue along EFPC contains few contaminants.

Some bird species which are potentially exposed are migratory species and are protected from harm under the Migratory Bird Treaty Act. Approximately 80 species occurring on the EFPC floodplain at some time during the year are considered migratory species. Table 6.61 lists these species. Thirty-four species are considered summer residents, migrating into the area in the spring, nesting and raising young, and departing in the fall. Fourteen species are winter residents, arriving in the fall, staying the winter, and departing in the spring. Thirty-two species are considered only transients, passing through the area only in the spring and fall, going further north in the spring and further south in the winter. As indicated earlier, the species receiving the greatest exposure would be those whose diets depend primarily on small invertebrates (i.e., primarily insects). Since this food source is more readily available during the spring, summer, and fall, the most exposed migratory bird species would be summer residents. Although transient species are also in the area during the spring and fall, individuals are in the area only for a few days and, therefore, would not consume a significant amount of contaminated food.

6.3.3.7 Aquatic and terrestrial adult arthropod survey

This section describes a survey of adult forms of aquatic and terrestrial arthropods at six sampling sites in the lower EFPC floodplain (Figs. 6.3 through 6.8) and one reference site on Hinds Creek (Fig. 6.9). The purpose of the survey was to characterize the taxonomic richness and composition, abundance, and diversity of the EFPC insect community. Arthropods are food for birds, mammals, amphibians, and other organisms.

Table 6.61 Migratory bird species occurring on the East Fork Poplar Creek floodplain[1]

Common name	Scientific name	Type of migrant[2]
Broad-winged Hawk	*Buteo platypterus*	SR
Yellow-billed Cuckoo	*Coccyzus americanus*	SR
Chuck-will's-widow	*Caprimulgus carolinensis*	SR
Whip-poor-will	*Caprimulgus vociferus*	SR
Common Nighthawk	*Chordeiles minor*	SR
Chimney Swift	*Chaetura pelagica*	SR
Ruby-throated Hummingbird	*Archilochus colubris*	SR
Yellow-bellied Sapsucker	*Sphyrapicus varius*	WR
Eastern Kingbird	*Tyrannus tyrannus*	SR
Great Crested Flycatcher	*Myiarchus crinitus*	SR
Acadian Flycatcher	*Empidonax virescens*	SR
Least Flycatcher	*Empidonax minimus*	T
Eastern Wood Pewee	*Contopus virens*	SR
Bank Swallow	*Riparia riparia*	T
N. Rough-winged Swallow	*Stelgidopteryx serripennis*	SR
Purple Martin	*Progne subis*	SR
Red-breasted Nuthatch	*Sitta canadensis*	WR
Brown Creeper	*Certhia americana*	WR
Winter Wren	*Troglodytes troglodytes*	WR
Gray Catbird	*Dumetella carolinensis*	SR
Wood Thrush	*Hylocichla mustelina*	SR
Hermit Thrush	*Catharus guttatus*	WR
Swainson's Thrush	*Catharus ustulatus*	T
Gray-cheeked Thrush	*Catharus minimus*	T
Veery	*Catharus fuscescens*	T
Blue-gray Gnatcatcher	*Polioptila caerulea*	SR
Golden-crowned Kinglet	*Regulus satrapa*	WR
Ruby-crowned Kinglet	*Regulus calendula*	WR

Table 6.61 (continued)

Common name	Scientific name	Type of migrant[2]
White-eyed Vireo	*Vireo griseus*	SR
Red-eyed Vireo	*Vireo olivaceus*	SR
Black-and-white Warbler	*Mniotilta varia*	SR
Prothonotary Warbler	*Protonotaria citria*	SR
Worm-eating Warbler	*Helmitheros vermivoros*	SR
Golden-winged Warbler	*Vermivora chrysoptera*	T
Blue-winged Warbler	*Vermivora pinus*	T
Tennessee Warbler	*Vermivora peregrina*	T
Orange-crowned Warbler	*Vermivora celata*	T
Nashville Warbler	*Vermivora ruficapilla*	T
Northern Parula	*Parula americana*	SR
Yellow Warbler	*Dendroica petechia*	SR
Magnolia Warbler	*Dendroica magnolia*	T
Cape May Warbler	*Dendroica tegrina*	T
Black-throated Green Warbler	*Dendroica virens*	T
Cerulean Warbler	*Dendroica cerulea*	SR
Blackburnian Warbler	*Dendroica fusca*	T
Yellow-throated Warbler	*Dendroica dominica*	T
Chestnut-sided Warbler	*Dendroica pensylvanica*	T
Bay-breasted Warbler	*Dendroica castanea*	T
Pine Warbler	*Dendroica pinus*	T
Prairie Warbler	*Dendroica discolor*	SR
Palm Warbler	*Dendroica palmarum*	T
Ovenbird	*Seiurus aurocapillus*	T
Louisiana Waterthrush	*Seiurus motacilla*	SR
Kentucky Warbler	*Oporornis formosus*	SR
Connecticut Warbler	*Oporornis agilis*	T
Mourning Warbler	*Oporornis philadelphia*	T

Table 6.61 (continued)

Common name	Scientific name	Type of migrant[2]
Common Yellowthroat	*Geothlypis trichas*	SR
Yellow-breasted Chat	*Icteria virens*	SR
Hooded Warbler	*Wilsonia citrina*	SR
Wilson's Warbler	*Wilsonia pusilla*	T
Canada Warbler	*Wilsonia canadensis*	T
American Redstart	*Setophaga ruticilla*	T
Bobolink	*Dolichonyx oryzivorus*	T
Orchard Oriole	*Icterus spurius*	SR
Baltimore Oriole	*Icterus galbula*	T
Scarlet Tanager	*Piranga olivacea*	T
Summer Tanager	*Piranga rubra*	SR
Rose-breasted Grosbeak	*Pheucticus ludovicianus*	T
Blue Grosbeak	*Guiraca caerulea*	T
Indigo Bunting	*Passerina cyanea*	SR
Purple Finch	*Carpodacus purpureus*	WR
Savannah Sparrow	*Passerculus sandwichensis*	WR
Vesper Sparrow	*Pooecetes gramineus*	T
Dark-eyed Junco	*Junco hyemalis*	WR
White-crowned Sparrow	*Zonotrichia leucophrys*	WR
White-throated Sparrow	*Zonotrichia albicollis*	WR
Fox Sparrow	*Passerella iliaca*	WR
Swamp Sparrow	*Melospiza georgiana*	WR

[1] Based on Howell and Monroe 1957, and Alsop 1971. This list has been edited to contain only those species which could be expected to occur on a regular basis on the EFPC floodplain.

[2] SR - Summer Resident
WR - Winter Resident
T - Transient (only occurring in the spring and fall)

Sampling methods

Sampling of adult arthropods was conducted using ultraviolet black-light traps at each of the seven sampling sites. Because the traps were left operating overnight unattended, each trap was sealed with custody tape. The lights were turned on at dusk. The following morning, the contents of each trap were emptied into chemically clean glass jars and subsequently enumerated. Each sample was labeled with site-specific data and sealed with custody tape. All traps were decontaminated prior to reuse. Following collection, the arthropods were separated by location of juvenile habitat (i.e., aquatic or terrestrial), keyed to family, and counted. The samples for each site were then composited and analyzed for body burden of contaminants of concern. Trapping took place for six nights at each of the six sample sites within the EFPC floodplain and for four nights at the Hinds Creek reference site. Due to equipment problems and to the small size of many of the samples collected on the fourth night of sampling, fourth-night samples were not analyzed for any of the seven sites.

Results

A total of 135 families from 19 arthropod orders were collected (Table 6.62). Three of the families belonged to 3 orders of the class Arachnida and the remaining 132 belonged to the class Insecta. Unidentified insects were included as a separate family under each order. Separation of the samples by location of juvenile origin (Tables 6.63 and 6.64) yielded 26 aquatic origin and 109 terrestrial arthropod families. Twenty-eight families were found at all sampling sites. Twelve aquatic families collected from EFPC were not found at the Hinds Creek reference site. Fifty-nine terrestrial families collected from EFPC were not found at Hinds Creek, and three were collected from Hinds Creek but not from EFPC.

The mean number of aquatic individuals per sample increased with sampling distance downstream from the Y-12 Plant, with the exception of a dramatic decrease in captures at Site 3 (Fig. 6.74). Site 3 is one of the two sites on EFPC having the greatest mercury contamination concentrations in soils. Site 6 had the greatest number of captures. Except for Site 3, all of the EFPC sites had more captures than did the Hinds Creek reference site. With the exception of Site 2, the mean number of terrestrial individuals per sample also increased with sampling distance downstream from the Y-12 Plant. Site 6 had the greatest number of captures, but fewer than the number of captures at the Hinds Creek reference site.

Table 6.62. Summary of EFPC adult aquatic and terrestrial arthropod species

List of Taxa	
Acari (Mites and Ticks) (T)	
Pseudoscorpionida (Pseudoscorpion) (T)	
Araneida (Spiders)	
	Araneidae (Orb Weaver Spider Family) (T)
Collembola (Springtails) (T)	
Ephemeroptera (Mayflies)	
	Baetidae (Small Minnor Mayfly Family) (A) Heptageniidae (Flatheaded Mayfly Family) (A) Ephemeridae (Common Burrower Mayfly Family) (A) Unidentified Ephemeroptera (A)
Odonota (Dragonflies and Damselflies)	
	Aeshnidae (Darner Family) (A) Libellulidae (Common Skimmer) (A) Lestidae (Stalked-winged Damselfly Family) (A)
Orthoptera (Grasshoppers, Crickets, and Katydids)	
	Tettigoniidae (Long-horned Grasshopper or Katydid Family) (T)* Gryllidae (Cricket Family) (T)
Dermaptera (Earwigs)	
	Labiidae (Little Earwing Family) (T)
Dictyoptera (Mantids and Cockroaches)	
	Mantidae (Mantids Family) (T)
Psocoptera (Booklice and Barklice)	
	Psocidae (Common Barklice Family) (T)* Unidentified Psocoptera (T)

Table 6.62. (continued)

List of Taxa	
Hemiptera (Bugs)	
	Cydnidae (Burrower Bug Family) (T) Pentatomidae (Stink Bug Family) (T) Coreidae (Squash Bug Family) (T) Lygaeidae (Seed Bug Family) (T) Reduviidae (Assassin and Thread-legged Bug Family) (T) Nabidae (Damsel Bug Family) (T) Miridae (Plant Bug Family) (T) Corixidae (Waterboatman Bug Family) (A)
Homoptera (Cicadas, Leafhoppers, Aphids, and Scale Insects)	
	Cixiidae (Cixiid Planthopper Family) (T) Delphacidae (Delphacid Planthopper Family) (T) Flatidae (Flatid Planthopper Family) (T)* Cercopidae (Spittle Bug Family) (T)* Cicadidae (Cicada Family) (T) Cicadellidae (Leafhopper Family) (T)* Membracidae (Treehopper Family) (T) Psyllidae (Jumping Plantlice Family) (T)* Aphididae (Aphid Family) (T)
Neuroptera (Dobsonflies, Lacewings, and Antlions)	
	Mantispidae (Mantislike Lacewing Family) (T) Hemerobiidae (Brown Lacewing Family) (T) Chrysopidae (Green Lacewing Family) (T)

Table 6.62. (continued)

List of Taxa	
Coleoptera (Beetles, Weevils, and Stylopids)	
	Carabidae (Ground Beetle Family) (T)*
	Dytiscidae (Predacious Diving Beetle Family) (A)
	Gyrinidae (Whirligig Beetle Family) (A)
	Hydrophilidae (Water Scavenger Beetle Family) (A)*
	Ptiliidae (Feather-winged Beetle Family) (T)
	Silphidae (Carrion Beetle Family) (T)
	Scaphidiidae (Shining Fungus Beetle Family) (T)
	Staphylinidae (Rove Beetle Family) (T)*
	Pselaphidae (Ant-loving Beetle Family) (T)
	Scarabaeidae (Lamellicorn Beetle Family) (T)
	Ptilodactylidae (Toed-winged Beetle Family) (T)
	Sandalidae (Cedar Beetle Family) (T)
	Heteroceridae (Variegated Mud-loving Beetle Family) (T)
	Elmidae (Drive or Riffle Beetle) (A)
	Limnichidae (Minute Marsh-loving Beetle Family) (A)
	Elateridae (Click Beetle Family) (T)
	Cantharidae (Soldier Beetle Family) (T)
	Throscidae (False Metallic Wood-boring Beetle Family) (T)
	Dermestidae (Skin Beetle and Larder Beetle Family) (T)
	Anobiidae (Drug-store and Death-watch Beetle Family) (T)
	Bostrichidae (Horned Powder-post Beetle Family) (T)
	Nitidulidae (Sap-feeding Beetle Family) (T)
	Rhizophagidae (Root-eating Beetle Family) (T)
	Cucujidae (Flat Bark Beetle Family) (T)
	Erotylidae (Pleasing Fungus Beetle Family) (T)
	Phalacridae (Shining Flower Beetle Family) (T)
	Coccinellidae (Ladybird Beetle Family) (T)
	Tenebrionidae (Darkling Beetle Family) (T)
	Mycetophagidae (Hairy Fungus Beetle Family) (T)
	Ciidae (Minute Marsh-loving Beetle Family) (T)
	Mordellidae (Tumbling Flower Beetle Family) (T)
	Cerambycidae (Longhorned Beetle Family) (T)
	Chrysomelidae (Leaf Beetle Family) (T)
	Bruchidae (Pea and Bean Beetle Family) (T)
	Platypodidae (Ambrosia Beetle Family) (T)
	Scolytidae (Bark Beetle Family) (T)*
	Curculionidae (Snout Beetle and True Weevil Family) (T)
	Unidentified Coleoptera (T)*

Table 6.62. (continued)

List of Taxa	
Hymenoptera (Wasps, Ants, and Bees)	
	Braconidae (Braconid Family) (T)* Ichneumonidae (Ichneumon Family) (T)* Unidentified Chalcidoidea (Parisitic Wasps) (T) Cynipidae (Gall Wasp Family) (T) Serphidae or Proctotrupidae (Serphid Wasp Family) (T) Formicidae (Ant Family) (T)* Vespidae (True Wasp Family) (T)* Sphecidae (Digger Wasp Family) (T) Unidentified Hymenoptera (T)
Trichoptera (Caddisflies)	
	Phryganeidae (Giant Casemaker Caddisfly Family) (A) Leptoceridae (Longhorned Casemaker Caddisfly Family) (A) Unidentified Trichoptera (A)*
Lepidoptēra (Moths, Butterflies, and Skippers)	
	Tineidae (Clothes Moth Family) (T) Psychidae (Bagworm Moth Family) (T) Gracillariidae (Gracillariid Moth Family) (T) Oecophoridae (Oecophorid Moth Family) (T)
	Plutellidae (Diamondback Moth Family) (T) Yponomeutidae (Ermine Moth Family) (T) Tortricidae (Tortricid Moth Family) (T)* Olethreutidae (Codling Moth Family) (T) Cochylidae (Cochylid Moth Family) (T) Pyralidae (Snout Moth Family) (T)* Pterophoridae (Plume Moth Family) (T) Geometridae (Geometer Moth Family) (T)* Lasiocampidae (Lappet Moth Family) (T) Sphingidae (Hawk Moth Family) (T) Notodontidae (Prominent Moth Family) (T)* Arctiidae (Tiger Moth Family) (T)* Lymantriidae (Tussock Moth Family) (T)* Noctuidae or Ctenuchidae (Noctuid Moth Family) (T)* Unidentified Lepidoptera (T)*
Mecoptera (Scorpionflies)	
	Panorpidae (Common Scorpionfly Family) (T)

Table 6.62. (continued)

List of Taxa	
Diptera (Flies)	
	Tipulidae (Crane Fly Family) (A)*
	Bibionidae (March Fly Family) (T)
	Mycetophilidae (Fungus Gnat Family) (T)
	Sciaridae (Dark-winged Fungus Gnat Family) (T)
	Cecidomyiidae (Gall Midge Family) (T)
	Dixidae (Dixid Fly Family) (A)
	Culicidae (Mosquito Family) (A)*
	Simuliidae (Black Fly Family) (A)
	Ceratopogonidae (Biting Midge Family) (T)
	Chironomidae (Midge Family) (A)*
	Tabanidae (Horse Fly and Deer Fly Family) (A)
	Stratiomyidae (Soldier Fly Family) (T)
	Empididae (Dance Fly Family) (A)
	Dolichopodidae (Long-legged Fly Family) (T)
	Phoridae (Humpbacked Fly Family) (T)
	Syrphidae (Flower Fly Family) (T)
	Lonchaeidae (Lonchaeid Fly Family) (T)
	Otitidae (Picture-winged Fly Family) (T)
	Tephritidae (Fruit Fly Family) (T)
	Piophilidae (Skipper Fly Family) (T)
	Sciomyzidae (Marsh Fly Family) (A)
	Lauxaniidae (Lauxaniid Fly Family) (T)
	Chamaemyiidae (Chamaemyiid Fly Family) (T)
	Ephydridae (Shore Fly Family) (A)
	Sarcophagidae (Flesh Fly Family) (T)
	Tachinidae (Parasitic Fly Family) (T)
	Unidentified Aquatic Diptera (A)*
	Unidentified Terrestrial Diptera (T)

(A) = Aquatic Origin.
(T) = Terrestrial Origin.
* = Found at all sites.

Table 6.63. Number of captured individuals of EFPC adult aquatic-origin arthropods by family

Taxon (family)	Sampling location						
	1	2	3	4	5	6	Hinds Creek
Aeshnidae				1	2		
Baetidae			230	237	619	494	14
Chironomidae	258	226	281	1030	2193	2006	733
Corixidae				16	131	567	
Culicidae	65	95	144	495	787	1637	203
Dixidae	46					161	
Dytiscidae		1			5	8	
Elmidae						2	
Empididae	1		1		20		49
Ephemeridae			2	11		16	87
Ephydridae		1					
Gyrinidae		1				4	
Heptageniidae	1	3	9	26	23	60	99
Hydrophilidae	38	9	2	19	17	24	7
Leptoceridae			1				
Lestidae				3			
Libellulidae						1	
Limnichidae					1		
Phryganeidae		3	1	1	4	10	61
Sciomyzidae		3		1	3		1
Simuliidae		1	64	17		294	1
Tabanidae						1	
Tipulidae	13	7	3	5	7	2	20
Unidentified Diptera	40	45	57	398	204	4717	20
Unidentified Ephemeroptera		2	1	28	21	1	45

Table 6.63. (continued)

Taxon (family)	Sampling location						
	1	2	3	4	5	6	Hinds Creek
Unidentified Trichoptera	4540	5110	51	1028	1182	761	304
Total individuals	5002	5507	847	3316	5219	10766	1644
Samples/site	5	5	5	5	5	5	3
Mean individuals/sample	1000	1101	169	663	1044	2153	548

Table 6.64. EFPC adult terrestrial-origin arthropods

Taxon[a]	Sampling location						
	1	2	3	4	5	6	Hinds Creek
Acari (Order)	1		1				
Anobiidae			2	5	1	11	
Aphididae		5	4		3		4
Araneidae	3	1	1				
Arctiidae	12	4	10	13	7	18	9
Bibionidae		30		1	128		
Bostrichidae	1	1	1		3		
Braconidae	11	16	10	24	5	7	4
Bruchidae							1
Cantharidae	1						
Carabidae	101	135	111	132	136	237	94
Cecidomyiidae			32	105	129	70	
Cerambycidae						1	
Ceratopogonidae		1		162	255		49
Cercopidae	3	20	2	13	49	43	6
Chamaemyiidae		1					
Chrysomelidae	2	16		1	3	3	1
Chrysopidae		1					
Cicadellidae	488	196	256	623	633	778	1547
Cicadidae		1			1		
Ciidae			1				
Cixiidae	3						
Coccinellidae		1					
Cochylidae	10		66				18
Collembola (Order)	1						
Coreidae		1					
Cucujidae						1	

Table 6.64. (continued)

Taxon[a]	Sampling location						
	1	2	3	4	5	6	Hinds Creek
Cuculionidae		11	1	1		2	
Cydnidae					6	3	2
Cynipidae		1					
Delphacidae	4		12			1	19
Dermestidae			1				
Dolichopodidae	22	1	12	5			50
Elateridae	2	2		5	1	5	2
Erotylidae	1	2		1	1		
Flatidae	39	31	235	212	281	166	162
Formicidae	25	57	4	7	14	46	50
Geometridae	46	101	72	98	147	149	125
Gracillariidae	1		3		1	2	
Gryllidae			17	2	7	9	3
Hemerobiidae	1			1			
Heteroceridae	7		4	2	318	38	308
Ichneumonidae	9	15	3	12	20	7	3
Labiidae	1			3		21	5
Lasiocampidae	6		1		5	5	
Lauxaniidae	1	5	1		1		
Lonchaeidae					20		
Lygaeidae	2			3			14
Lymantriidae	7	8	6	9	8	13	7
Mantidae							1
Mantispidae							1
Membracidae	1					1	
Miridae			4	13	5		11

Table 6.64. (continued)

Taxon[a]	Sampling location						
	1	2	3	4	5	6	Hinds Creek
Mordellidae	1			5	1		3
Mycetophagidae						3	
Mycetophilidae			1				
Nabidae			1	1	2	1	4
Nitidulidae		4	1	2	1	2	22
Noctuidae	88	26	131	157	202	189	203
Notodontidae	17	36	35	22	24	34	25
Oecophoridae	2		3		1	3	
Olethreutidae	1		1	1			
Otitidae	4	1	1	1	1	2	
Panorpidae	1			2			
Pentatomidae					1		
Phalacridae	1						
Phoridae			12				
Piophilidae					1		
Platypodidae	1	1			21	2	2
Plutellidae			15				
Proctotrupidae		2			2		
Pselaphidae	1		2				
Pseudoscorpionida (Order)			1				
Psocidae	6	2	3	7	5	1	14
Psychidae			2			16	
Psyllidae	13	13	2	20	32	5	23
Pterophoridae		1	1	1	1	1	
Ptiliidae					1	2	

Table 6.64. (continued)

Taxon[a]	Sampling location						
	1	2	3	4	5	6	Hinds Creek
Ptilodactylidae				1			
Pyralidae	85	42	85	148	229	183	125
Reduviidae				3	1	2	1
Rhizophagidae					1	2	
Sandalidae	1						
Sarcophagidae				1	1	1	1
Scaphidiidae	1	1					
Scarabaeidae		6	2	55	2	1	2
Sciaridae					74	64	
Scolytidae	4	10	1	2	17	19	6
Silphidae		1	1		2	4	1
Sphecidae	3	7				4	
Sphingidae	1		1				
Staphilinidae	144	101	216	108	188	590	287
Stratiomyidae		1					
Syrphidae				7		3	1
Tachinidae	3	1		15	1	18	5
Tenebrionidae	2	1	1	4		3	
Tephritidae			2				
Tettigoniidae	1	9	3	12	5	1	3
Throscidae	1						
Tineidae			2				
Tortricidae	32	8	49	61	57	36	36
Unidentified Calcidoidea		1					
Unidentified Coleoptera	35	28	35	28	112	317	161

Table 6.64. (continued)

Taxon[a]	Sampling location						
	1	2	3	4	5	6	Hinds Creek
Unidentified Diptera	20		3				5
Unidentified Hymenoptera	19	8	24	21	30	71	
Unidentified Lepidoptera	191	128	410	541	771	656	316
Unidentified Psocoptera			4				
Vespidae	24	15	7	42	12	128	1
Yponomeutidae	1	1		2	1		2
Total individuals	1516	1119	1931	2723	3989	4001	3745
Samples/site	5	5	5	5	5	5	3
Mean individuals/sample	303	224	386	545	798	800	1248

[a]Unless noted otherwise, taxon is family.

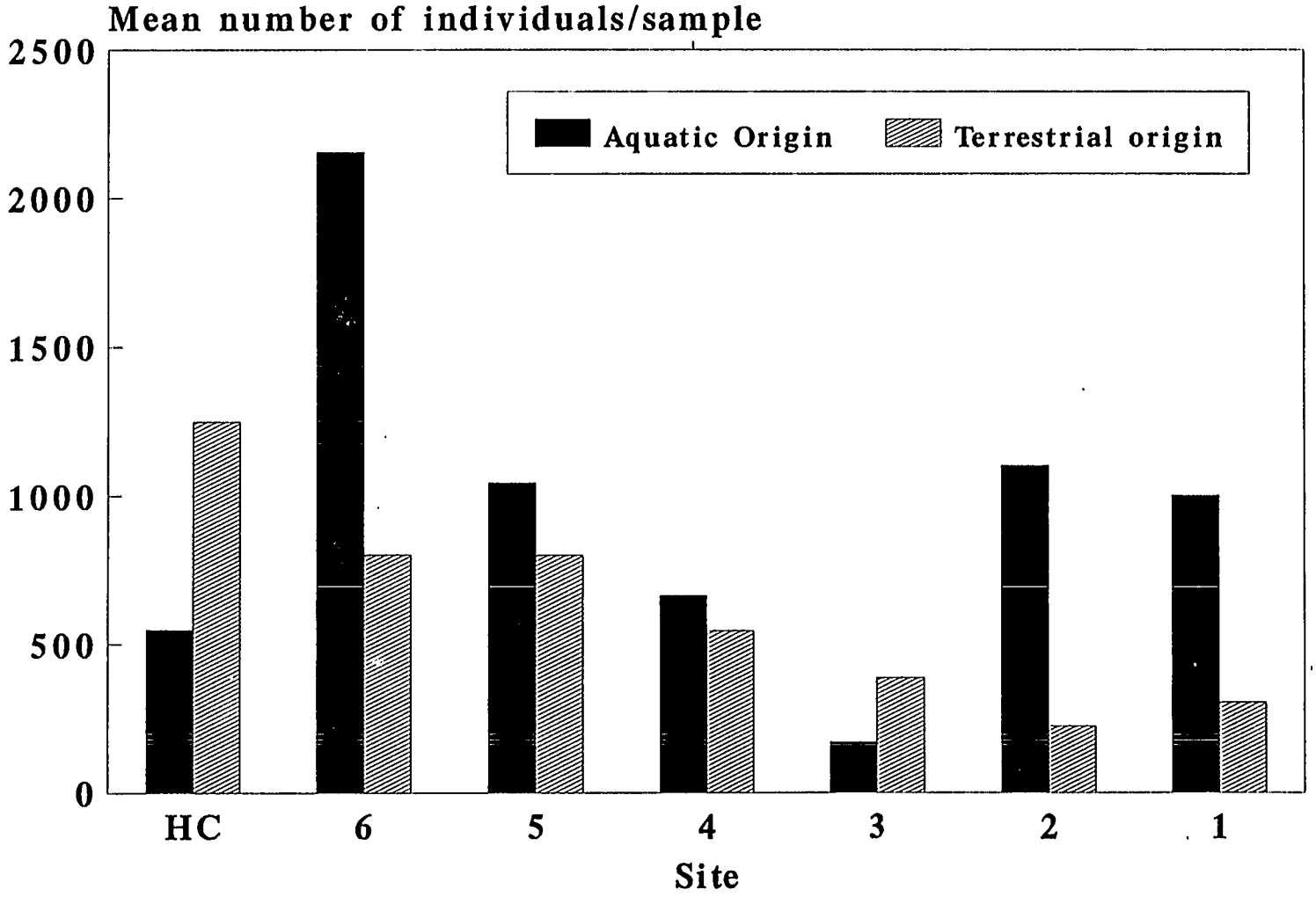

Fig. 6.74. Mean number of adult aquatic and terrestrial arthropods per sample at EFPC and Hinds Creek sampling sites.

The ratio of aquatic to terrestrial arthropods (A:T) varied from a low of 1:2.3 at Site 3 and at the Hinds Creek reference site to a high of 5:1 at Site 2 (Table 6.65). The ratio of aquatic-origin to terrestrial-origin arthropods was greater at all sites except at Site 3 and at the Hinds Creek reference site. The A:T ratio increased at Site 2, decreased significantly at Site 3, increased at Site 4, was the same at Site 5, and increased again at Site 6. The sharp dropoff of aquatic-origin arthropods is the reason for the decrease of the A:T ratio at Site 3, but at the Hinds Creek reference site the decrease of the A:T ratio is attributable more to a substantial increase in terrestrial-origin arthropods rather than to a major decrease in aquatic-origin arthropods.

Taxonomic richness for each sample site was determined by counting the number of families per site. Taxonomic richness exhibited a distinct longitudinal gradient along the EFPC sampling sites (Fig. 6.75). Richness was lowest at Site 1 and increased with distance from the Y-12 Plant, reaching a maximum at Site 6. Richness at the Hinds Creek reference site was greater than at all EFPC sites.

Diversities (H′) of aquatic arthropods were similar at the two EFPC sites farthest upstream (Sites 1 and 2) and also at the remaining four downstream sites (Sites 3, 4, 5, and 6). The diversity of aquatic arthropods at all the EFPC sites was less than that for the Hinds Creek reference site (Table 6.65). The diversity of terrestrial arthropods at EFPC was similar at all sites and higher than at the Hinds Creek reference site.

For Sites 1 and 2, Unidentified Trichoptera with aquatic juveniles was the dominant taxon (at 91% and 93%, respectively). Given the benthic macroinvertebrate data, the Unidentified Trichoptera are probably Hydropsychidae. At Sites 3, 4, and 5 and at the Hinds Creek reference site, Chironomidae were found in greatest relative abundance (at 33%, 31%, 42%, and 45%, respectively). Unidentified Diptera were most abundant at Site 6 (at 44%). Cicadellidae was the terrestrial taxon found in greatest relative abundance at all sites (at 32%, 18%, 23%, 19%, and 41%, respectively) except at Site 3 and Site 5, where Unidentified Lepidoptera was the most abundant (at 21% and 19%, respectively).

Limitations and uncertainties

A major limitation on this survey and the interpretation of the sampling data is that it represents a brief sampling duration. Time and funding limits restricted the experimental design to a simplified, unreplicated survey that could be completed in a matter of days. This type of survey does not permit observation of seasonal variations that certainly occur in the EFPC ecosystem. In addition, short- or long-term trends cannot be determined unless comparison can

Table 6.65. Summary of EFPC adult aquatic and terrestrial arthropod sampling

Measurement	Sampling location						
	1 (n=5)	2 (n=5)	3 (n=5)	4 (n=5)	5 (n=5)	6 (n=5)	Hinds Creek (n=3)
Mean number of aquatic-origin individuals/sample	1000	1101	169	663	1044	2153	548
Mean number of terrestrial-origin individuals/sample	303	224	386	545	798	800	1248
Ratio (A:T)[a]	1:0.3	1:0.2	1:2.3	1:0.8	1:0.8	1:0.4	1:2.3
Taxonomic richness[b]	27	28	29	30	33	34	37
Diversity index[c] (A)	0.63	0.51	2.51	2.39	2.27	2.42	2.54
Diversity index (T)	3.77	4.21	3.90	3.83	4.02	3.85	3.33

[a]A = Aquatic; T = Terrestrial.
[b]Mean number of taxa collected in quantitative samples.
[c]Shannon-Wiener Diversity Index.

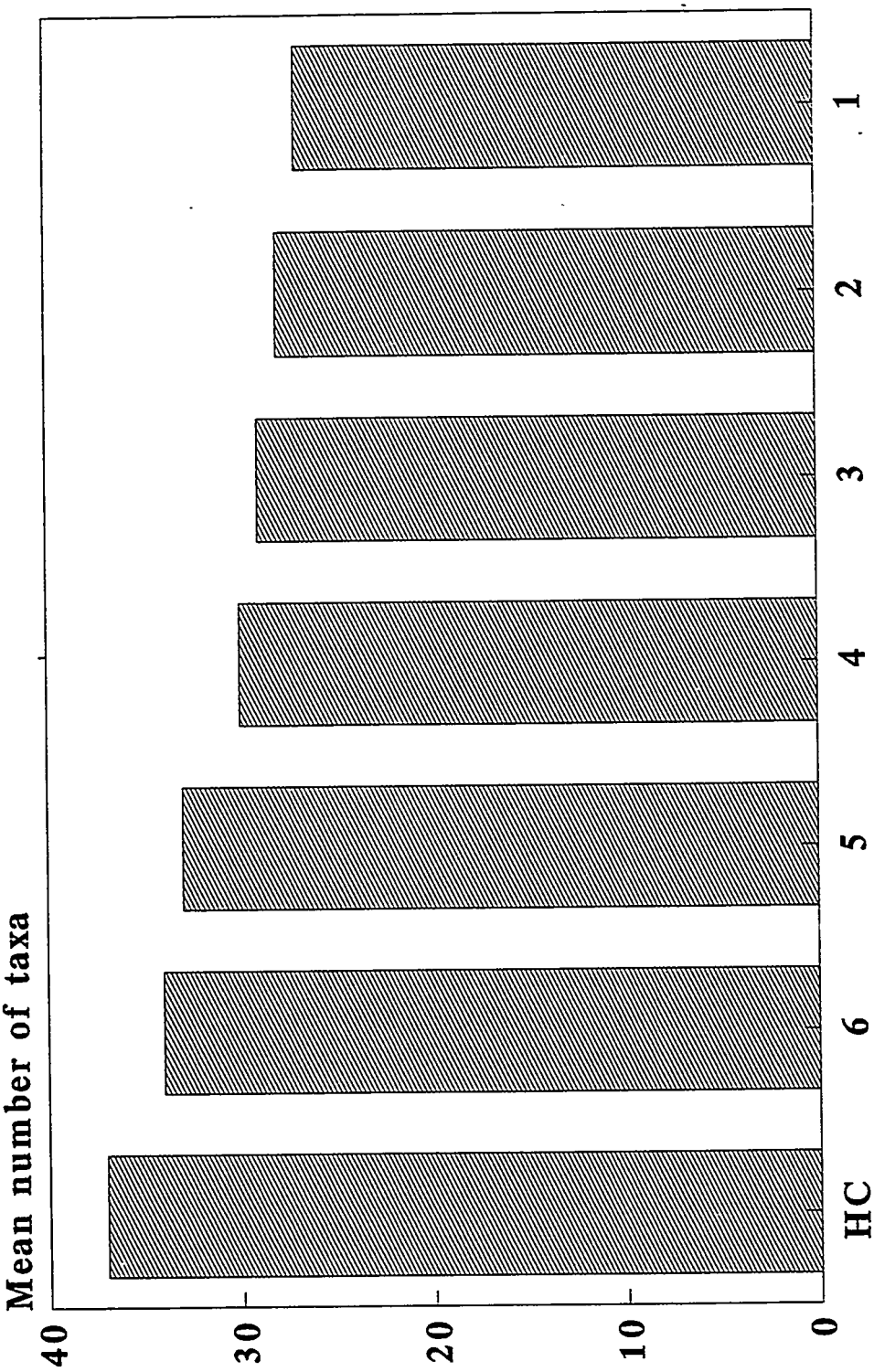

Fig. 6.75. Mean taxonomic richness for adult arthropods at EFPC and Hinds Creek sampling sites.

be made to existing historical data. Unfortunately, no historical survey data on the aquatic and terrestrial adult arthropod community along EFPC are known to be available.

Equipment malfunction and loss resulted in fewer numbers of samples at the Hinds Creek reference site (i.e., 5 samples per site on EFPC versus 3 for Hinds Creek). This makes comparison between EFPC sites and the Hinds Creek reference site difficult.

A further source of uncertainty is the dissimilarity between Site 3 and the other sites with respect to disturbances and light intensity. Site 3 is located adjacent to a heavily travelled road and directly across from a shopping center with several mercury vapor lights. The remainder of the EFPC sites and the Hinds Creek reference site are generally isolated from heavy traffic and bright light sources. What effect the heavy traffic and additional light sources may have had on the success of adult arthropod capture at Site 3 is unknown.

6.3.3.8 Earthworm survey

An investigation of earthworms—another indicator species—was conducted to complement the arthropod study within the EFPC floodplain. A recent ORNL study (Talmage and Walton 1990) investigated the occurrence of contaminants in earthworms (*Lumbricus terrestris*) from limited locations within the EFPC floodplain. Although data from this study are not yet published, preliminary results indicate that certain contaminants are above background levels.

Sampling methods

Earthworms were sampled using a 25 cm (10 in.) square and 20 cm (8 in.) deep sampling frame. This surface-area-to-volume ratio is appropriate for earthworm population sampling (Zicsi 1962). Twelve samples were collected per site. Each site was sampled within the small mammal sampling grid, using trap stations as sampling points. Samples were obtained by driving the welded steel sampling frame into the soil to the appropriate depth. The block of soil was then lifted and placed on plastic sheeting for hand-sorting (Nelson and Satchell 1962).

A number of environmental factors can influence earthworm populations and distribution: soil pH, moisture content, temperature, soil type, food supply, aeration and carbon dioxide concentration, and organic matter. Of these factors, soil pH is most likely to generate population variations among sites within the EFPC floodplain. Many species have been shown to be acid intolerant (below 5.0), having a slightly acidic optimum pH of 6.0. The mean soil pH values of horizon 1 at Sites 2 and 3 are 6.4 and 7.2, respectively. At present, these are the only sites for which pH data are available.

Results

Sites 1, 2, and 5 had similar mean number of individuals per sample—roughly 40% of the numbers exhibited at Sites 3, 4, and 6 (Table 6.66, Fig. 6.76). Sample numbers at the former sites averaged 60% above the Mill Branch reference site. Site 3 generated the highest mean number of individuals per sample (196 individuals/m^2); this was the only site that supported significant grass cover.

Contaminant concentrations

Results for average concentrations of arsenic, chromium, mercury, and zinc in the soil at each of the six survey sites along EFPC are summarized in Sect. 3. Earthworm abundances show no relationship to concentrations of arsenic, chromium, or zinc. The mercury concentrations in soil and earthworms and earthworm abundance are depicted in Fig. 6.76.

Conclusions

The means of earthworm samples taken along EFPC, when compared with average soil contaminant concentrations, yield no observable correlations. Differences in the abundance of earthworms at the sites may be attributed to differences in environmental factors among the sites other than the contaminants of concern. Comparison of sample means with available soil pH values, soil types, and soil conditions provided no additional insights to the observed population levels and distributions.

6.3.3.9 Terrestrial vegetation survey

This section describes a survey of vegetation at the six sampling sites in the EFPC floodplain and one site on Mill Branch, a tributary watershed. The Mill Branch site was used as a reference site for terrestrial fauna; its limitations as a reference site for vegetation in the EFPC floodplain are discussed below.

The purpose of the survey was twofold. The first was to evaluate potential explanations of differences of fauna among the sites. Habitat differences may account for differences in animal populations among sites; similarities in habitat may suggest that variability in animal populations is related to differences in other factors, such as contaminant concentration and/or distribution. The second purpose of the vegetation survey was to investigate potential direct effects of contaminants on the vegetation. Because site replication was not possible, a field survey had little power to determine effects related to perturbation.

Table 6.66. Abundance, species richness, species diversity, and species evenness of earthworms along EFPC

Parameter	Sampling location						
	Site 1	Site 2	Site 3	Site 4	Site 5	Site 6	Mill Branch
Abundance (individuals/m^2)	62	68	196	146	72	167	40
SR[a]	2	2	4	3	3	2	3
H'[b]	0.66	0.66	1.37	1.04	1.04	0.66	1.04
J[c]	0.95	0.95	0.99	0.95	0.95	0.95	0.95

[a]SR = species richness.
[b]H' = species diversity.
[c]J = species evenness = H'/H'max.

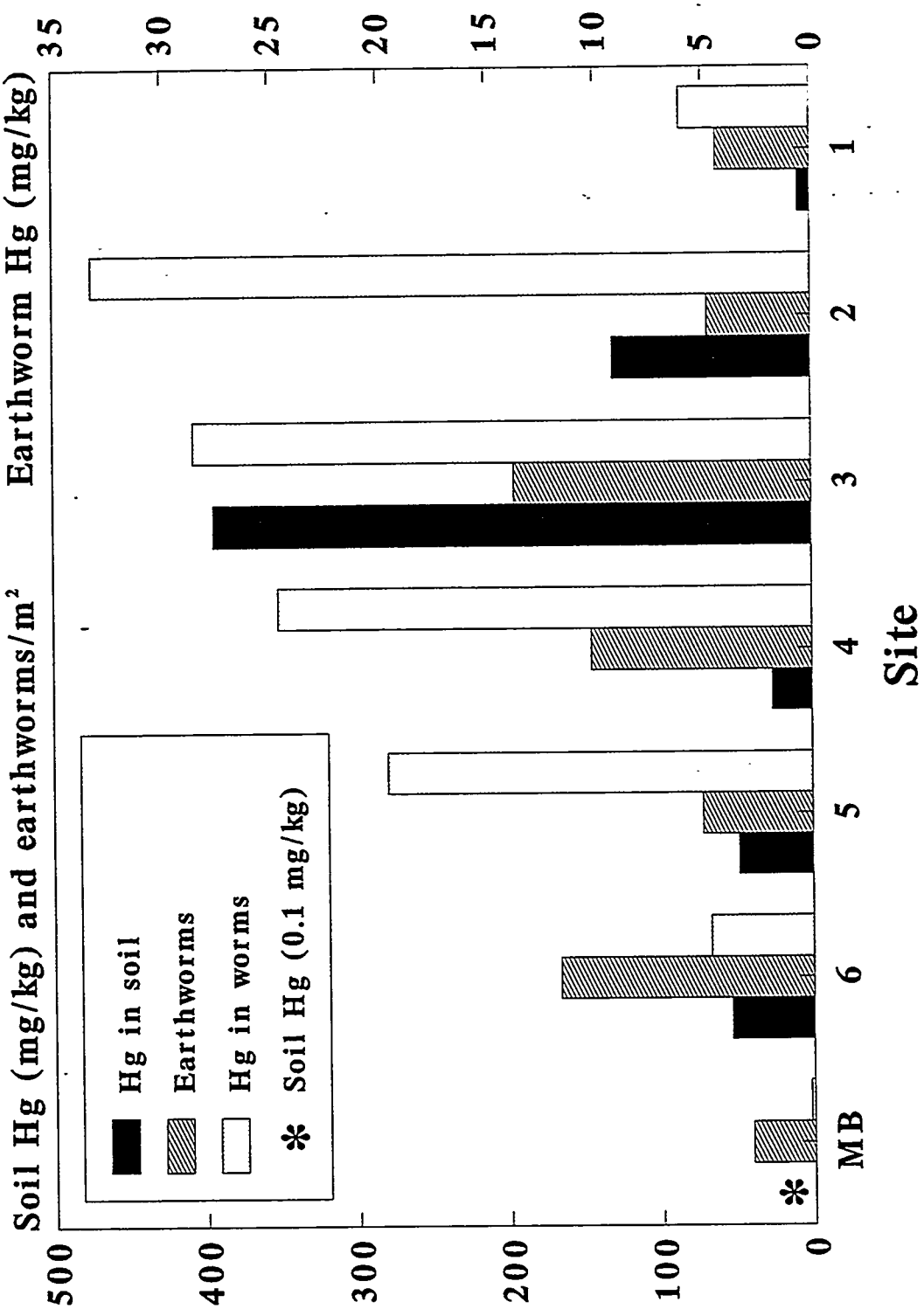

Fig. 6.76. Earthworm numbers at EFPC and Mill Branch sampling sites.

Direct effects of contaminants on vegetation could be as dramatic as species die-back or as subtle as a shift in dominance related to contaminant load. A shift in dominance could result from species' differing tolerances to contaminants at various stages of their life history. Mercury is reported to have an inhibitory effect on germination and early growth of some plant species (Hsu and Chou 1992; Renjini and Janardhanan 1989; Maury et al. 1986; Spencer and Siegel 1978; and Siegel 1977). One way to detect a potential shift in dominance is to quantify recruitment of the dominant species. Shifts in dominance are natural during ecological succession; however, a shift in dominance away from climax species may indicate significant disturbance.

Sampling methods

Vegetation was surveyed using point-intercept transects and belt transects. Percent cover was calculated as the number of survey points along a line transect where a given species was recorded either above or below the point. The sum of percent cover of all species encountered during sampling may exceed 100 because more than one species may be recorded at each point; vegetation is typically stratified (e.g., canopy, subcanopy, shrub layer, and ground cover). Relative cover is calculated by dividing the number of times a species is encountered along a transect by the total number of encounters of all species. The sum of relative cover for all species is ~100. Abundance is the number of individuals of species in a 2-m (6.6-ft) wide belt transect centered lengthwise on the line transect used to estimate cover. In this study, abundance of trees and shrubs only was determined. Relative abundance is abundance divided by the sum of abundance of all species. Density is abundance divided by the area sampled. Species richness is the total number of species recorded. The cover types used in habitat mapping for EFPC are presented in Sect. 6.2. A measure of the similarity among sites was determined and used to better describe the sites and to facilitate use of the habitat data for comparison of animal populations among the sites. Sorenson's Index was used as a measure of similarity:

$$S_s = \frac{2(\text{number of species in common})}{2\,(\text{number of species in common}) + \text{number of species in area 1} + \text{number of species in area}}$$

Sorenson's Index ranges in value from 0 to 1; a value of 1 denotes complete similarity in terms of species composition.

Results

A summary of dominant species and the contribution of each to relative cover and percent of cover for each sample location are shown in Table 6.67. The relative abundance of each tree

Table 6.67. Summary of dominant canopy species of terrestrial vegetation at EFPC and Mill Branch

Dominant canopy species	Relative cover	Percent cover	Relative abundance (tree species)
Site 1			
Cercis canadensis	17.1	56	16.9
Platanus occidentalis	11.4	30	—
Fraxinus pennsylvanica	9.3	28	18.5
Ulmus rubra	6.4	20	4.6
Acer negundo	—	—	32.3
Prunus serotina	—	—	9.2
Site 2			
Carpinus caroliniana	9.1	29	25.7
Acer negundo	9.1	29	4.4
Ulmus rubra	6.7	22	2.7
Platanus occidentalis	6.7	22	0.9
Fraxinus pennsylvanica	—	—	33.6
Prunus serotina	—	—	8.0
Acer saccharum	6.2	20	4.4
Site 3A			
Acer negundo	40.3	96	73.3
Fraxinus pennsylvanica	6.0	14	26.7
Salix nigra	6.0	14	—
Site 3B			
Aster sp.	29.6	64	
Glecoma hederacea	11.1	24	
Fescue	9.3	20	
Verbesina sp.	9.3	20	
Acer negundo	—	—	85.7
Salix nigra	—	—	8.6
Acer rubrum	—	—	5.7

Table 6.67. (continued)

Dominant canopy species	Relative cover	Percent cover	Relative abundance (tree species)
Site 4			
Acer negundo	28.1	75	69.6
Platanus occidentalis	17.7	47	0.9
Ulmus rubra	7.3	19	0.9
Celtis occidentalis	—	—	15.2
Fraxinus pennsylvanica	—	—	9.8
Site 5			
Acer negundo	32.2	88	70.0
Platanus occidentalis	12.6	34	10.0
Fraxinus pennsylvanica	9.2	25	—
Ulmus rubra	—	—	10.0
Celtis occidentalis	—	—	10.0
Site 6			
Acer negundo	31.1	88	94.3
Platanus occidentalis	14.4	41	—
Fraxinus pennsylvanica	17.8	50	3.8
Celtis occidentalis	—	—	3.8
Mill Branch			
Liquidambar styraciflua	17.2	66	1.3
Pinus virginana	14.8	56	0.9
Cornus florida	13.1	50	22.6
Acer saccharum	—	—	22.1
Acer rubrum	—	—	16.4
Corylus americana	—	—	16.8

species is also indicated. Site 3 consisted of two distinct habitats—a bottomland hardwood forest and an old field, labelled 3A and 3B, respectively. It is interesting to note that at Sites 3A, 4, 5, and 6, boxelder (*Acer negundo*) had the greatest percent cover, relative cover, and relative abundance. Site 1 is dominated by redbud (*Cercis canadensis)* in terms of percent cover and relative cover; however, boxelder stems are twice as numerous. More than 50% of the tree seedlings at Site 1 are boxelder. Assuming stable or climax conditions in this part of the floodplain, boxelder will most likely dominate the site in the future. Site 2 is dominated by ironwood (*Carpinus caroliniana*) and boxelder in terms of percent cover and relative cover; however, green ash (*Fraxinus pennsylvanica*) is more abundant and comprises greater than 50% of tree seedlings present. Barring further disturbance, Site 2 may undergo a shift in dominance from ironwood and boxelder to ash. Site 7 on Mill Branch is dominated by sweetgum (*Liquidambar styraciflua*), Virginia pine (*Pinus virginiana*), and dogwood (*Cornus florida*). Dogwood and three other tree species—sugar maple (*Acer saccharum*), red maple (*Acer rubrum*), and hazelnut (*Corylus americana*)—are the most abundant on the site. This site is apparently in a state of ecological succession toward a more mesic community.

Species richness is greater in forest habitats at a younger seral stage than are the bottomland hardwood sites (Table 6.68). Species richness at younger forest habitat Sites 1, 2 and 7 was 45, 49, and 42, respectively. Species richness on bottomland hardwood (Sites 3A, 4, 5, and 6) was relatively low and ranged from 20 to 30. This pattern of increasing species richness as forests develop is typical of forests in the eastern United States that are comparable in topographic complexity.

Density of woody species at the seven sites varied considerably (Fig. 6.77). Yet no pattern in density related to distance from Lake Reality or known contaminant load is apparent. The density at Site 7 was more than double that of the other sites and the resultant compaction and disruption of the soil profile can result in slow recovery of the forest floor vegetation long after grazing ceases. Closure of the canopy by the large trees present at Site 3 results in low light intensity at the forest floor. These conditions together may account for the paucity of tree seedlings at Site 3. The contribution of juveniles to the measure of density is of interest. The fact that many young trees are present at most sites indicates that tree seedlings of at least some species are becoming established. For the bottomland hardwood forest sites, the most abundant trees seedlings—boxelder—are those of the canopy dominant. This suggests that, relative to the other species present, contaminants are not inhibiting seedling establishment of the dominant species in the community.

Table 6.68. Species richness of terrestrial vegetation at EFPC and Mill Branch

Parameter	Sampling location							
	1	2	3A	3B	4	5	6	Mill Branch
Total species richness	45	49	20	20	27	30	22	42
Species richness of trees	12	13	2	3	8	4	3	21

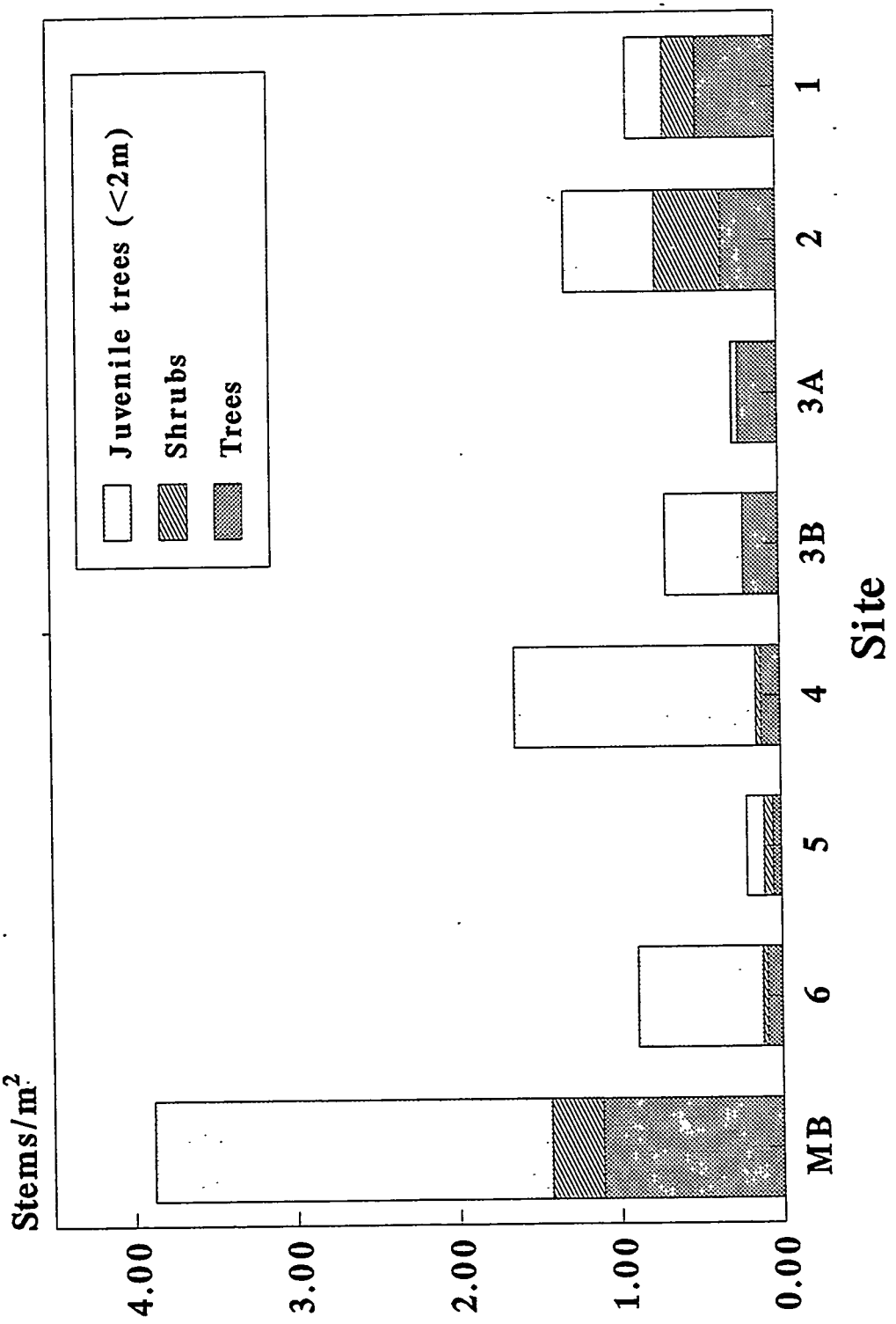

Fig. 6.77. Density of woody species at EFPC sampling sites.

For most sites, non-juvenile trees are less than 55% of the stems per square meter, but for Site 3A, the community is made of 86% non-juvenile trees. There is relatively little recruitment at Site 3A; juveniles make up only 14% of the total density compared to a range of 25 to 91% for the other sites (Table 6.69). The low level of recruitment of trees at Site 3A cannot be explained by a larger population of seed-eating rodents at the site; the rodent population sampled at that site was smaller than at most other EFPC sites sampled. If the relatively high level of contaminants at Site 3A is influencing germination or establishment of trees, then it would be expected that recruitment of trees in the adjacent old field habitat, Site 3B, would be similarly reduced. Recruitment of trees is not reduced in that habitat. Available information indicates that the site has not been grazed in the past eighteen years but was grazed by cattle for a period of about twelve years (1962 - 1974).

Another hypothesis for the observed pattern at Site 3 is that the contaminants have a synergetic effect with light intensity that results in reduced germination of seeds or seedling establishment of the component tree species. If this effect exists, then one would expect to see some evidence of the same pattern in reduction of recruitment at Site 2—a site with similar levels of contamination—assuming that Site 2 has a similar level of light intensity on the forest floor. Overall, recruitment is not reduced at Site 2 relative to the other sites, but recruitment of boxelder is low given that boxelder is one of the dominant species at that site. The data cannot rule out the possibility that contaminants are a factor in germination or in establishment of boxelder under low-light (closed-canopy) conditions; however, it is more plausible that past grazing coupled with low light intensity are the significant contributors to the lack of juvenile trees.

Table 6.70 shows a matrix of Sorenson's Index of similarity values among sites and levels of similarity among sites; and Table 6.71 shows those sites to which each site is most similar and least similar. Site 3B, an old field habitat, was not included in the analysis; all other sites are forested. Site 3B is not expected to bear any similarity to the other sites.

With the exception of Site 3A, the bottomland hardwood sites (4, 5, and 6) are most similar to each other. Although Site 3A exhibits the physical characteristics of a bottomland hardwood site (i.e., broad floodplain and deep alluvial soil) and supports a mature forest of typical bottomland hardwood species, the herb layer and tree seedlings were sparse. The site most similar to Site 3A was Site 2; however, the level of similarity (0.21) was less than for any other pairs of most similar sites.

Table 6.69. Percentage of density of woody species at EFPC and Mill Branch, contributed by trees, shrubs, and juvenile trees

Woody species (type)	Sampling location							
	1	2	3A	3B	4	5	6	Mill Branch
Trees (%)	53	26	86	31	7	24	10	28
Shrubs (%)	22	31	0	0	2	24	3	8
Juvenile trees (%)	25	43	14	69	91	52	87	64

Table 6.70. Similarity of plant species composition at EFPC and Mill Branch site

Sites	1	2	3A	4	5	6	Mill Branch
1	1	0.30	0.22	0.20	0.24	0.19	0.26
2	0.30	1	0.26	0.28	0.32	0.22	0.31
3A	0.22	0.26	1	0.18	0.24	0.25	0.11
4	0.20	0.28	0.18	1	0.40	0.31	0.24
5	0.24	0.32	0.24	0.40	1	0.35	0.22
6	0.19	0.22	0.25	0.31	0.35	0	0.18
Mill Branch	0.26	0.31	0.11	0.24	0.22	0.18	1

Table 6.71. Matrix of similarity of terrestrial vegetation sampling sites at EFPC and Mill Branch

Parameter	Sampling location						
	1	2	3A	4	5	6	Mill Branch
Most similar to site — (measure of similarity	2 (0.27)	5 (0.32)	2 (0.21)	5 (0.50)	4 (0.50)	5 (0.50)	2 (0.28)
Least similar to site — (measure of similarity)	4,6 0.14	6 (0.28)	7 (0.07)	3A (0.12)	7 (0.16)	7 (0.12)	3A (0.07)

Mill Branch shows relatively little similarity to bottomland hardwood sites on EFPC (Table 6.71). This site is an immature upland forest in the process of change in dominance. Species richness is high, particularly for tree species. Therefore, Mill Branch is not considered a reference area for plant communities on the EFPC floodplain because of the differences in physical characteristics (e.g., soil type and hydrologic regime) of the site compared to the other sites. Therefore, although the EFPC sites are dissimilar to the reference site in terms of species composition, contaminants in the EFPC floodplain are not considered to be a primary cause of the noted differences.

Limitations and uncertainties

The uncertainties in the results of the survey of terrestrial vegetation on the EFPC floodplain and Mill Branch are related to temporal distribution of the samples and errors in measurement. The surveys were conducted during the late summer and early fall of one growing season; thus, winter annuals and spring ephemerals are underrepresented or absent in the survey. A survey during one growing season represents a single "snapshot" of the vegetation, and natural variability in abundance of annuals or browsed herbs is not reflected in the results. In addition, the methods for measuring percent cover involved visual projection from a line transect up into the forest canopy. This method has a moderate potential for error; thus, this measure should not be considered absolute. Consistent methods were used for all sites, so the results can be considered comparable across sites. It is important to state again that the Mill Branch site is not a suitable reference site for the terrestrial vegetation of the EFPC floodplain from the composition view. No suitable reference areas were found in the surrounding area; floodplains similar to EFPC in physical characteristics are in agricultural use, primarily in pasture.

6.3.3.10 Sewer Line Beltway

Contaminated soil from the EFPC floodplain was used as topsoil for parts of the Sewer Line Beltway (SLB) (Sect. 3.1.2.2). For that reason, the SLB has been included within the scope of this RI. Therefore, it was necessary to assess the risks to the ecosystem from contaminants in EFPC floodplain soils deposited in the SLB. Because portions of the SLB within the EFPC floodplain were not evaluated separately from the floodplain, the following discussion concerns only portions of the SLB outside of the floodplain.

Aquatic biota

No aquatic habitats border the SLB outside the EFPC floodplain (Map 12), so exposure of contaminants to aquatic biota in the SLB is not significant.

Terrestrial vegetation and habitats

By virtue of its size, location, and function, the SLB does not provide important habitat for terrestrial vegetation. Vegetation typical of areas disturbed by construction and other usual urban activities is present and does not appear to be adversely affected by soil contaminants.

Terrestrial animals

Because the total area of the SLB is small, the fraction of a typical terrestrial animal's home range affected by SLB contaminants is small. In addition, levels of soil contaminants are low in most of the SLB (Map 12). Therefore, it is highly unlikely that toxic effects of exposures to SLB soils have caused or will cause reductions in terrestrial animal populations or in their ability to maintain themselves.

6.3.3.11 Protected species and habitats

Protected species and significant habitats are those defined and governed by the Endangered Species Act and Executive Orders 11988 (Protection of Floodplains) and 11990 (Protection of Wetlands). Potential harm to protected species and habitats must be considered in an ERA because the value placed on these resources by the public has been demonstrated by enactment of federal laws intended to preserve them or promote their conservation. State and local laws governing wildlife protection must be considered for the same reasons. States prepare lists and rankings of rare species for which protection is either recommended or required by law. Thus, listed species and their critical habitat are logical assessment endpoints in an ERA.

A number of rare species listed by the state of Tennessee as endangered, threatened, or of special concern/in need of management are known to occur on ORR. Four plant species, *Aureolaria patula, Cimicifuga rubifolia, Delphinium exaltatum,* and *Juglans cinerea,* are candidates for listing by the U.S. Fish and Wildlife Service (Eisler).

Protected terrestrial plants

A brief description of protected plant species known to occur on ORR and potentially occurring on the EFPC floodplain based on the presence of suitable habitat follows.

Spreading false-foxglove (*Aureolaria patula*) — This member of the snapdragon family is a large, showy-flowered perennial that is narrow in range and rare within its range. The species is currently listed as a candidate for federal listing as threatened. The category of current listing is C1, which means that formal listing is expected based on information to date concerning its

rarity. It is present on the Clinch River in the Oak Ridge area and is present on Poplar Creek, into which EFPC flows. Suitable habitat is limestone outcrops or rocky banks along a creek or river.

Pink lady-slipper (*Cypripedium acaule*) — Pink lady-slipper is a showy, pink-flowered perennial that is prized in the horticulture trade. The species is listed as endangered in the state based on evidence of large numbers being taken from the wild and lack of commercial success with propagation. Its habitat is moist to dry woods.

Golden seal (*Hydrastis canadensis*) — Golden seal is a fairly widely distributed perennial species but is relatively rare within its range because of collection for its reputed medicinal value. It is found primarily in rich moist woods. It is listed as threatened in Tennessee.

Butternut (*Juglans cinerea*) — Butternut is similar to the common black walnut and grows in rich woods. Butternut is listed as threatened in Tennessee and is a candidate for federal listing as threatened. The category of current listing is C2, which means that more information is needed to determine whether it should be proposed for formal listing.

Canada lily (*Lilium canadense*) — Canada lily grows in moist to wet lowlands, typically at the edge of woods. It is listed as threatened in Tennessee.

Ginseng (*Panax quinquifolium*) — Ginseng is widely distributed in Tennessee but is heavily collected from the wild for its reputed medicinal value. It is typically found in rich woods.

Purple fringeless orchid (*Platanthera peramoena*) — This member of the orchid family grows in open, wet habitat (e.g., wet meadows). It is listed as threatened in Tennessee.

Carey's saxifrage (*Saxifraga careyana*) — Small basal rosettes of this species grow in limestone outcrops on bluffs along the Clinch River. It is listed as of special concern in Tennessee.

Lesser ladies-tresses (*Spiranthes ovalis*) — This is typically a diminutive orchid on the ORR, with only one of two basal leaves each season. It flowers in early fall and grows in moist to dry woodland.

A survey of the EFPC floodplain was conducted to determine the presence of rare plant species. No protected species were observed.

Protected terrestrial vertebrates

Federally- and state-protected terrestrial vertebrate species designated as threatened or endangered and that occur or for which suitable habitat exists within the EFPC floodplain are discussed briefly below.

Indiana Bat (*Myotis sodalis*) — In the warmer months of the year, Indiana bats are widely dispersed in suitable habitat throughout a large portion of their range. The Indiana bat hibernates in caves during the winter and raises its young in maternal colonies during the warmer months. The preferred areas for these maternity colonies are hardwood forests along streams. The Indiana Bat is listed as Federally Endangered. A field sampling of bats on EFPC failed to find individuals of this species (Harvey 1992).

Gray Bat (*Myotis grisescens)* - Gray bats are cave residents year-round, although different caves are usually occupied in summer and winter. They forage primarily over water along rivers or lake shores. The gray bat is listed as endangered by the FWS. No individuals of this species were captured during the sampling for bats on EFPC (Harvey 1992).

Bachman's Sparrow (*N n*) — Typically occurring in open pine stands with heavy ground cover, in old fields, in open wooded pasture, and in very young pine plantations, this once common species is now rare and local within the state in spite of available suitable habitat. The last recorded observation of this species near EFPC was of two singing individuals on ORR in 1982. The Bachman's sparrow is listed as State Endangered.

Northern Harrier (*N n*) — An uncommon-to-rare migrant/winter resident, this harrier frequents weedy or grassy open fields throughout the state. This species has been observed nearby in habitats similar to open fields along EFPC. It is listed as State Threatened.

Cooper's Hawk (*Accipiter cooperii*) — A very secretive permanent resident of dense forest throughout the state. Uncommon to rare as a breeding bird, it has been sighted several times during the breeding season on ORR. The Cooper's hawk is listed as State Threatened.

Sharp-shinned Hawk (*Accipiter striatus*) — A rare permanent resident that may be expected in every county in Tennessee, it prefers dense stands of conifers for nesting. It has not been recorded on ORR during the breeding season but has been observed in the surrounding area. The sharp-shinned hawk is listed as State Threatened.

Bewick's Wren (*N n*) — Formerly very common within the state, this permanent resident suffered severe population declines for unknown reasons and now is locally uncommon to rare. It occurs

most frequently in rural areas, often near old homesites, farms, or residences having nearby grassy areas, gardens, hedgerows, and thickets. This wren has not been recorded recently on ORR. Suitable habitat may exist within the privately-owned areas of the EFPC floodplain. The Bewick's wren is listed as State Threatened.

Grasshopper Sparrow (*Ammodramus savannarum*) — Formerly a common summer resident in grassy or weedy fields throughout the state, its populations are much reduced and are extirpated from some former breeding areas that still have suitable habitat. This sparrow has been recorded in the past during the breeding season on ORR in idle pasture with tall grasses. It is listed as State Threatened.

The Tennessee Wildlife Resources Agency has designated several species as "Wildlife in Need of Management." Species within this category known to occur on or near EFPC are listed below.

Black-crowned Night Heron (*Nycticorax nycticorax*): known to occur along Clinch River.

Black Vulture (*Coragyps atratus*): known to occur along EFPC.

Red-shouldered Hawk (*Buteo lineatus*): known to occur on ORR.

Common Barn Owl (*Tyto alba*): known to occur on ORR.

Protected aquatic species

Tennessee Dace (*Phoxinus tennessensis*): known to occur on ORR.

Yellow-bellied Sapsucker (*N n*): secondary observation (drilled trees) in floodplain (winter visitor).

Southeastern Shrew (*N n*): known to occur on ORR.

Protected habitats

Currently there are no administratively protected habitats on ORR. Protection of wetlands is to be considered in decision-making on actions by DOE (10 CFR 1022 implementing regulations for Executive Order 11990). As many as 2.6 ha (6.43 acres) of wetlands in the EFPC floodplain may be contaminated with mercury and possibly other COCs. Proposed actions in wetlands by DOE must comply with provisions of the Clean Water Act, Section 404, and associated wetlands assessment and NEPA requirements.

Critical habitat of federally-listed species is administratively protected. Although there is not designated critical habitat in the EFPC study area, impact to habitat potentially used by threatened and endangered species could constitute an impact to the species and must be evaluated. For example, spreading false-foxglove (*Aureolaria patula*) is a Category 1 candidate for federal listing and apparently suitable habitat is present along the streambank of EFPC. As noted in preceding paragraphs of this section, surveys for bat and rare plants failed to find these species present in EFPC floodplain.

6.3.4 Summary of Effects Assessment

Data on the toxicity of inorganic and organic mercury compounds to fish demonstrate a positive relationship between dose and effect. Toxicity tests on a variety of aquatic and terrestrial organisms using EFPC surface water, soils, or sediments have shown deleterious effects, and in two cases, the effects were greatest at the sites nearest the Y-12 Plant. The results of surveys of indicator organisms at EFPC are also indicative of effects from exposure to contaminants released from the Y-12 Plant. The abundance of fish and flying insects and the taxonomic richness and diversity of fish, flying insects, and benthic invertebrates generally and EPT families particularly, increased with increasing distance from the Y-12 Plant, whereas the number of species of fish classified as tolerant of degraded water quality, and the body burdens of Aroclor 1260 in fish, crayfish, and flying insects and mercury concentrations in fish, crayfish, and earthworms and their gut contents showed the opposite trend. Compared to a reference stream, EFPC exhibited fewer species of fish, families of benthic macroinvertebrates, EPT families, mean number of flying insects with terrestrial juveniles, lower taxonomic diversity and evenness for fish, benthic macroinvertebrates, and flying insects with aquatic juveniles. Also, compared to a reference stream, EFPC exhibited higher concentrations of mercury in fish, crayfish, and benthic macroinvertebrates, earthworms and their gut contents, in flying insects with aquatic juveniles (5 sites), those with terrestrial juveniles (two sites), and Aroclor 1260 in fish, crayfish, and flying insects with terrestrial juveniles at all sites. Two Carolina wrens, a heron's liver and feathers, mice, shrews, a vole, grass, browse, and garden vegetables from the EFPC floodplain had elevated concentrations of contaminants (i.e., mercury, Aroclor 1260, or pesticides). Together, there are many ecological effects to organisms living in the aquatic and terrestrial habitats.

A fall survey of birds identified 43 species on the EFPC floodplain but effects on resident or transient individuals and population densities or community structure of residents (year round or seasonal) could not be determined. On the basis of limited vegetation sampling, including the browse and garden study, there appear to be effects of exposure to EFPC contaminants on plant

communities. The SLB has no demonstrated exposure pathways to EFPC biota. The threatened and endangered survey, both literature and field, revealed no species requiring protection of their populations, nor of their support populations (food webs) and habitats.

6.4 RISK CHARACTERIZATION

According to the *Framework for Ecological Risk Assessment* (EPA 1992a), the ERA consists of four interrelated activities—problem formulation, exposure characterization, effects characterization, and risk characterization (see Sect. 6.1.2 for description). Section 6.4 deals with risk characterization, which aggregates the effects of exposure and stressor response on indicator organisms, summarizes risk according to the weight-of-evidence approach, and interprets the ecological significance of these findings.

This ERA takes two interrelated approaches to ecological risk characterization for EFPC and its floodplain. The first is the application of the quotient method (Barnthouse et al. 1986) to various criteria for protection of ecological resources. In this method, observations are compared to numerical criteria for protection of ecological resources (e.g., assessment endpoints). The ratio of the observed contaminant concentration to the protection criterion indicates the severity of impact or risk, with larger quotients indicating larger potential impacts or risks. With sufficiently large data sets, statistical analyses can be applied to the observed values, or data distributions can be compared to criterion distributions to determine the degree of overlap. Because of the uncertainty surrounding both sampling data and the criteria themselves, this approach requires careful interpretation of quotients near 1, but it provides a clear indication of high (10 or greater) or low (<0.1) risks. Quotients are also used as input to the second approach.

The second interrelated approach is to apply weight-of-evidence arguments to evaluate the strength of the relationship between stressors and observed effects on indicator species and the implications for assessment endpoints. The principal reason for using a weight-of-evidence approach is that there are multiple lines of evidence that must be evaluated. This method helps to identify causes of observed ecological responses, using arguments derived from human epidemiology. In this approach, a causal relationship between a stressor and a response is proposed. Then a series of questions or criteria is applied to the proposition. Not all criteria need be satisfied to demonstrate that the proposition is true, but weight is added to a conclusion by each criterion that is satisfied in the proposition(s). Ultimately, professional judgement is used to establish the strength of the causal relationship. The weight-of-evidence approach is especially

useful when: (1) there are insufficient data for robust statistical analyses, (2) toxicity or other criteria are uncertain, or (3) exposure models are not sufficiently precise for statistical hypothesis testing.

The criteria in the weight-of-evidence approach used in this ERA are as follows:

- Temporal association—did the proposed cause precede the effect?
- Spatial association—is the affected population exposed to the proposed causative agent?
- Stressor response—does the severity of the effect vary in response to the magnitude of exposure to the proposed causative agent?
- Strength of association—are there other potential causes that could be present or act antagonistically/synergistically to produce the observed effect?
- Plausibility—does the proposition make sense and is it consistent with known etiological and scientific principles? Is there a reasonable mechanism of action?

Each of these criteria is further explained below.

Temporal associations may be difficult to demonstrate. If measures of biological populations or physical media were made before and after the proposed cause acted, a temporal relationship may be easily shown. If measurements were not made before the proposed cause, as is often the case, there may be no direct evidence for temporal association. Correlated fluctuations in the proposed stressors and the effect can provide evidence for both temporal association and quantitative stressor response.

Spatial association may be demonstrated by a decrease in the severity of effect in the indicator organisms with distance from the proposed causative agent. It may also be shown by a distribution of effects in relation to contaminant transport, such as location in the surface water of a stream, in a groundwater plume, or downwind from an airborne source. Chemical transport models may describe the spatial association in quantitative or qualitative terms. Spatial association can also be demonstrated through comparisons of stressed situations relative to an unstressed reference situation.

A positive correlation between the magnitudes of the stress and the response is strong evidence for causality. If a contaminant can be measured in the exposure media, then it can be

quantitatively compared to the severity of observed effects. Body burden measurements are useful in establishing the realness of stressor/response relationships. Otherwise, indirect measures of the stress may be made, including expected attenuation with distance from the proposed source and temporal relatedness of fluctuations in the source and the affected environmental resource by an indicator population and habitat.

Demonstrating strength of association requires an adequate data base and application of good scientific judgement. Confounding factors must be taken into account in evaluating the strength of association. For example, several contaminants may be released into exposure media, and a population may respond to one or more of them, simultaneously. Alternatively, if stressors that act antagonistically are released from the source at different times, fluctuations in the response may reflect fluctuations in both the causative agent and its antagonist. The presence of an antagonist may mask the effects of a stressor, weakening the apparent temporal associations between stressor and effect.

Scenarios by which the stressor can cause the observed response must be plausible. Scientifically sound principles, preferably backed by experimental evidence or other field observations, must be used in developing and evaluating the plausibility, or reasonability, of the proposition.

These criteria are evaluated quantitatively or qualitatively, depending on the types and quality of data available. Thus, a gradient of body burdens in indicator organisms with distance from the proposed source may be used as evidence for spatial association, whereas evaluation of a temporal association may be based on circumstantial evidence rather than on data obtained before and after the event. Experimental evidence may also be used to evaluate these and other weight-of-evidence criteria.

6.4.1 Risk Integration for Current Conditions

This subsection compares effects observed under current conditions with criteria for the protection of fish and wildlife, and it presents weight-of-evidence arguments bearing on the causality of ecological effects in EFPC and its floodplain. It describes degradation of aquatic communities in EFPC as a result of waterborne contaminants being released from the Y-12 Plant, and it evaluates the impacts of contaminants in EFPC floodplain soils on terrestrial biota.

6.4.1.1 Comparison of effects with criteria

Risks to biota can be characterized by comparing exposure concentrations to specific criteria intended to be protective of the organism or its function in the ecosystem. Thus, criteria may

be established to limit the concentrations of chemicals in surface water in order to protect aquatic biota (TDEC 1987) or limits may be set on contaminant concentrations in tissues in order to protect organisms or their predators. Criteria may also be established by modeling the transfer of contaminants from exposure media through the food chain to the ultimate biotic receptors, thereby taking into account bioaccumulation of contaminants. In the quotient method, if the ratio of exposure concentration to criterion concentration is sufficiently less than 1, risk is considered not to exist, whereas if the quotient is sufficiently larger than 1, risk is inferred (Barnthouse et al. 1986). Expert scientific judgment must be used to interpret quotients near 1. This method requires consideration of the uncertainty of estimating both the exposure concentration and the criterion values.

Ambient Water Quality Criteria (AWQC) for freshwater aquatic life. Assessment endpoint 5a (Sect. 6.1) compares concentrations of contaminants of concern in water to AWQCs for the protection of freshwater biota. AWQCs take into account bioconcentration of contaminants from water into the tissues of aquatic organisms. Because bioconcentration of lipophilic organic chemicals is usually high, the criteria for these compounds are often below the analytical detection limit; the same is true for mercury in surface water.

Organic analytes. Concentrations of PCBs, pesticides, and PAHs—other analytes of concern—were all below detection limits in surface water. Therefore, the AWQC cannot be assessed directly for these analytes. However, aquatic biota bioconcentrate organic contaminants from water at rates that are both chemical-specific and taxa-specific. Published bioconcentration factors (BCFs) can be used to back-calculate water concentrations that would theoretically correspond to observed body burdens. That approach is followed in the subsections below for organic analytes with endpoints below analytical detection limits.

Inorganic analytes. Table 6.72 lists AWQCs for contaminants of concern, the observed surface water concentrations of these contaminants (in grab samples collected during biota sampling), and the quotient of the two values when the quotient could be calculated. The results show that the concentrations of mercury in surface water exceeded AWQCs at Sites 1, 2, and 3 by a large margin. At the remainder of the sites, mercury concentrations were below detection limits. Since the criterion is also below detection limits, the quotient cannot be complete and, therefore, is not evaluated for these samples.

AWQCs for arsenic, chromium, copper, nickel, and zinc were not exceeded. The AWQC for lead (3.0 μg/L) was exceeded by a factor of 1.03 at Sites 1 and 3. Further sampling would

Table 6.72. Comparison of surface water contaminant concentrations (μg/L) to AWQCs for inorganics

Sample	Analytes							
	Arsenic	Cadmium	Chromium	Copper	Lead	Mercury	Nickel	Zinc
AWQC[a]	190	1.0[b]	11[c]	12[b]	3.0[b]	.012	.160[b]	106[b]
Site 1, observed	<5	<5	<4	<6	3.1	0.54	<8	33.4
quotient	<0.03	<5	<.4	<0.5	1.03	**45**	<0.05	0.32
Site 2, observed	<5	<5	<4	<6	<2	0.43	<8	19.5
quotient	<0.03	<5	<0.4	<0.5	<0.7	**36**	<0.05	0.18
Site 3, observed	<5	<5	<4	<6	3.1	0.32	<8	14.5
quotient	<0.03	<5	<0.4	<0.5	<1.03	**27**	<0.05	0.14
Site 4, observed	<5	<5	<4	<6	<2	<0.2	<8	33.7
quotient	<0.03	<5	<0.4	<0.5	<0.7	<17	<0.05	0.32
Site 5, observed	<5	<5	<4	<6	2.9	<0.2	<8	24.1
quotient	<0.03	<5	<0.4	<0.5	1.0	<17	<0.05	0.23
Site 6, observed	<5	<5	<4	<6	2.4	<0.2	<8	15.3
quotient	<0.03	<5	<0.4	<0.5	0.8	<17	<0.05	0.14
Hinds Creek reference, observed	<5	<5	<4	<6	<2	<0.2	<8	6.1
quotient	<0.03	<5	<0.4	<0.5	<0.7	<17	<0.05	0.06

[a] Water quality criteria for protection of freshwater biota (TDEC Chap. 1200-4-3).
[b] Depends on hardness of water.
[c] Value for chromium^{6+}.

be required to validate the exceedance by lead and justify remedial actions to reduce lead levels to the AWQC.

PCBs. The AWQC for chronic exposure of freshwater biota to PCBs is 0.001 μg/L (Table 4.1). PCB was not detected in surface water samples taken during biota sampling (Sect. 6.2.3.2). However, the contract-required detection limit for PCB in water is 0.5 μg/L; at the contract-required detection limit, undetected concentrations of PCB in surface water could exceed the AWQC by a factor of 500. The accumulation of PCB in fish and crayfish shown in Sect. 6.2.3.2 indicates that PCBs are present in EFPC and must be monitored by further surveillance of aquatic biota.

BCFs for Aroclor 1254 have been reported to be in the range of 49,000 L/kg to approximately 100,000 L/kg; a value of 194,000 was reported for Aroclor 1260 (Lyman et al. 1990). Using the latter value, the concentration of PCB in surface water corresponding to fish body burdens of Aroclor 1260 at Site 1 can be calculated:

$$C_{water} = C_{biota}/BCF$$

$$C_{water} = 2600\ \mu g/kg\ /\ 194{,}000\ L/kg\ \text{(redbreast sunfish)}$$
$$= 0.013\ \mu g/L$$

This calculated value exceeds the AWQC of 0.001 μg/L by a factor of 13.

Chlordane. Chlordane was chosen as representative of the pesticides since it caused the highest pesticide body burden in stonerollers and redbreast sunfish. Chlordane has been shown to adversely effect sensitive species of fish and aquatic invertebrates at nominal water concentrations between 0.2 and 3.0 μg/L (Eisler 1990).

Pesticides were not detected in surface water samples taken during the biota sampling (Section 6.2.3.2) at the contract-required detection limit of 0.5 μg/L. Chlordane, dieldrin, and heptachlor were detected in whole-body samples of redbreast sunfish (maximum of approximately 60 μg/kg) and stonerollers (maximum of approximately 100 μg/kg for chlordane and dieldrin, approximately 45 μg/kg for heptachlor). In crayfish chlordane, dieldrin, and DDE were detected at a maximum of approximately 10 μg/kg.

No AWQC for chlordane has been set. Although not an ARAR, the recommended freshwater aquatic life protection criterion is 0.0043 μg/L (24-h average) (Eisler 1990). The

concentration of chlordane in surface water corresponding to the maximum body burden of 100 μg/kg can be calculated from a BCF of 37,800 (Lyman et al. 1990):

$$C_{water} = C_{biota}/BCF$$

$$C_{water} = 100\ \mu g/kg / 37{,}800\ L/kg \text{ (redbreast sunfish)}$$
$$= 0.0026\ \mu g/L$$

This result is less than the recommended chronic criterion.

PAHs. PAHs were not detected in surface water samples taken during biota sampling (Sect. 6.2.3.2). AWQCs for PAHs have not been established, and no criteria for protection of fish and aquatic life are listed as ARARs for PAHs. The most stringent ARAR for PAHs in water is a proposed maximum contaminant level (MCL) of 0.03 μg/L for chrysene and pyrene (Table 4.1). Although EFPC is not being used as a drinking water source, it ultimately flows into a drinking water reservoir, so the MCL will be evaluated as an endpoint.

At the contract-required detection limit of 10 μg/L in water, undetected concentrations of PAH analytes in water could exceed the MCL by a factor of 300.

Accumulation of PAHs was observed in EFPC aquatic biota samples. The maximum concentrations of chrysene, benzo(a)anthracene, and benzo(a)pyrene observed in crayfish were 20-25 μg/kg (Fig. 6.53) and levels from 18 μg benzo(a)anthracene/kg to 52 μg chrysene/kg were observed in stonerollers (Fig. 6.48).

BCFs for organic chemicals have been shown to be related to their octanol-water partition coefficients (K_{ow}) by the relationship:

$$\log BCF = 0.76 \log K_{ow} - 0.23$$

[derived from data for 84 compounds, $r^2 = 0.82$ (Veith et al. 1980)]. BCFs for PAHs ranging from approximately 300 to approximately 115,000 have been calculated from their octanol-water partition coefficients (K_{ow}). As an example, using the observed maximum of 18 μg benzo(a)anthracene/kg in stonerollers, and the reported BCF for benzo(a)anthracene of 11,700, the benzo(a)anthracene concentration in the water column corresponding to the observed body burdens was calculated:

$C_{water} = C_{biota}/BCF$
$= 18\ \mu g/kg / 11{,}700\ L/kg$
$= 0.0015\ \mu g/L$

Therefore, the MCL for benzo(a)anthracene (0.1 μg/L) appears not to be exceeded in surface water in EFPC.

Similar calculations for other selected PAHs are presented in Table 6.73. None of the PAHs whose concentrations were reported in aquatic biota appear to exceed ARARs in EFPC surface water.

Sediment exposures. Benthic invertebrates may be exposed to contaminants leached from sediments into pore water. The AWQC is assumed to apply to pore water, since it is designed to be protective of aquatic biota. Pore water concentrations were calculated from mean and maximum sediment concentrations of mercury and PCBs (Sect. 6.2.2.1); they were 0.017 and 0.11 μg mercury/L and 0.016 and 0.025 μg PCB/L. An exposure quotient can be calculated from these values:

$$EQ = C_{pore\ water} / AWQC$$

The calculated pore water exposure quotients were as follows:

for mercury, $EQ_{mean} = 0.017/0.012 = 1.4$
$EQ_{max} = 0.11/0.012 = 9.2$

for PCBs, $EQ_{mean} = 0.016/0.001 = 16$
$EQ_{max} = 0.025/0.001 = 25$

Therefore, benthic invertebrates are potentially at risk from exposure to contaminants in sediments, especially to PCBs.

Soil ingestion exposure. Potential toxicity by incidental ingestion of soil can occur, especially to small mammals that burrow and subsequently groom themselves and/or that ingest soil-dwelling prey (e.g., shrews). Feeding studies have shown that absorption of mercury and other metals by laboratory mice from contaminated soil taken from the EFPC floodplain caused no discernible toxicological effects during a 20-month exposure (Revis et al. 1989). In this study, the fraction of soil in the diet was 0.05; higher exposures under natural conditions are very

Table 6.73. Calculation of apparent concentrations of PAHs in surface water

Compound	ARAR		Maximum observed in redbreast sunfish (μg/kg)	Calculated BCF[a]	Calculated maximum concentration in water (μg/L)
	Domestic water supply	Recreation			
Benzo[a]anthracene	0.1	0.3	5.5	11,700	0.0005
Benzo[a]pyrene	0.2	0.3	3.0	28,200	0.0001
Benzo[b]fluoranthene	0.2		5.7	28,200	0.0002
Benzo[g,h,i]perylene			3.7	68,200	0.00005
Benzo[k]fluoranthene	0.2		5.9	28,200	0.0002
Chrysene	0.2	0.03	7.4	11,700	0.0006
Dibenzo[a,h]anthracene	0.3		5.6	113,000	0.00005
Pyrene		0.03	10.0	2,800	0.0036

[a] Calculated from K_{ow}

unlikely. Therefore, it is concluded that incidental ingestion of soil does not present a risk to terrestrial biota in the EFPC floodplain.

A method for estimating the potential intake of soil by shrews or other small animals at a similar trophic level was presented in Sect. 6.2.2.1, where daily intake of a contaminant in soil was estimated as $0.004 \times C_{soil}$. The exposure quotient can be calculated as:

$$EQ = 0.004 \times C_{soil} / CD$$

where CD, the comparison dose, is the toxicological benchmark to which the organism is to be protected. It should preferably be a Lowest Observable Effects Level, No Observed Adverse Effects Level, or similar measure of effect for susceptible species.

The comparison dose values used for this assessment are:

PCB, 0.005 mg/kg (Eisler 1986)
Chlordane, 0.1 mg/kg [most conservative value reported (Eisler 1990)]
PAHs, 30 mg/kg (Eisler 1987)

Exposure quotients were calculated using the highest concentrations of soil contaminants observed in EFPC floodplain soils (Table 3.31). The results were:

PCB (maximum 3.8 mg/kg soil): $EQ = 0.004 \times 3.8 / 0.005 = 3.0$
Chlordane (maximum 0.024 mg/kg soil): $EQ = 0.004 \times 0.024 / 0.1 = 0.001$
PAH (maximum 10 mg/kg soil): $EQ = 0.004 \times 10 / 30 = 0.001$

These calculations show that incidental ingestion of chlordane and PAHs in soil is not a risk to small mammals or birds. The calculated ingestion of PCB at the maximum observed soil concentration is higher than benchmark values for PCB ingestion. Therefore, soil-ingesting biota such as shrews and birds may be at risk from PCBs in EFPC floodplain soil.

FDA action limits. Although FDA action limits are based on human health, this is important in the protection of fishery resources. The action limit protects the fish populations as an ecological resource per se and, also, as fish that are consumed. The values used in this analysis were 1 mg mercury/kg (49 FR 45663), 0.3 mg chlordane/kg (55 FR 14359), and 2 mg PCBs/kg (49 FR 21514) body weight. Redbreast sunfish and stonerollers sampled at Sites 1, 2, and 3 exceeded the criterion for mercury. PCB levels in redbreast sunfish and stonerollers

exceeded FDA limits at Sites 1 and 2, and in sunfish at Site 3. Mercury levels in crayfish exceeded FDA limits at Sites 1 and 2, although no crayfish samples exceeded action limits for PCBs. None of the aquatic biota exceeded the FDA action limit for chlordane.

Toxicological effects. Assessment endpoints 5b and 7b (Sect. 6.1) compare body burdens of contaminants in aquatic and terrestrial biota to concentrations that have been proposed to be protective against toxicological effects in the indicator organisms.

Mercury. Table 6.74 lists these criteria for mercury and summarizes the instances in which they were exceeded in sampled EFPC biota. A criterion level of 5 mg mercury/kg (whole body) was proposed for freshwater fish by the FWS (Eisler 1987a) based on toxic effects in brook trout, which died after exposures that caused an accumulation of 5 to 7 mg mercury/kg body weight (EPA 1980, 1985). Although this species is not indigenous to or present in EFPC, the proposed criterion would likely provide a conservative benchmark value for the measurement endpoint associated with assessment endpoint 5b, thereby providing protection of aquatic biota from harm from accumulated contaminants. The criterion level was exceeded in 2 of 7 crayfish samples and in 1 of 10 composite stoneroller samples (Table 6.74).

The limit for residual mercury in mammalian tissues was based on observations of toxicity in organisms with concentrations in brain, kidney, blood, and hair greater than 1.1 mg mercury/kg tissue (Eisler 1987). One mouse, sampled at Site 2, had a body burden of 1.1 mg/kg, one mouse sampled in the EFPC floodplain had a level about half as high, and the remainder had undetectable levels of mercury ($< \sim 0.4$ mg/kg). Shrews had body burdens considerably above the criterion (1.1.mg/kg), at 1.9 and 7.9 mg/kg. It is not clear that animals with whole-body burdens of <2 mg mercury/kg have concentrations in brain, kidney, blood, and hair in excess of this criterion, but the criterion is exceeded sufficiently in the shrew with 7.9 mg mercury/kg to suggest the possibility of toxic effects.

No whole-body criterion for mercury in birds was found; however, the proposed criterion for mercury in feathers was 5 mg/kg (Eisler 1987). One sample of feathers from a heron from Site 5 had a concentration of 5.3 mg mercury/kg, slightly above the criterion. The mercury concentration in the heron's liver was nearly 9 mg/kg, far above the criterion for mammalian organs. Whole-body concentrations of mercury in wrens ($n=2$) were approximately 3.5 mg/kg, also above the criterion for mammals.

Table 6.74. Comparison of body burdens to criteria for toxicological effects from exposure to mercury[a]

Organism, analysis	Criteria (mg/kg)	Sampling sites 1	2	3	4	5	6	Reference
Aquatic[a]								
Stoneroller, whole body[b]	5	3.1	6.4	1.9	0.7	0.7	0.4	<0.2
quotient		0.6	1.3	0.4	0.1	0.1	0.1	<0.04
Redbreast sunfish, whole body	5	1.1	1.1	1.9	0.9	0.5	0.2	<0.2
quotient		0.2	0.2	0.4	0.2	0.1	0.04	<0.04
Crayfish, whole body	5	6	3.3	0.7	—[c]	—	0.2	<0.2
quotient		1.2	0.7	0.1			0.04	<0.04
Heron, feathers	5	—	—	—	—	5.5	—	—
quotient						1.1		
Heron, liver	(1.1)[d]	—	—	—	—	9.0	—	—
quotient						8.2		
Terrestrial[e]								
Mouse, whole body	1.1	<0.3	0.9	<0.4	<0.2	<0.3	—	<0.4
quotient		<0.3	0.8	<0.4	<0.2	<0.3		<0.4
Shrew, whole body	1.1	—	—	—	1.9	7.9	—	—
quotient					1.7	7.2		
Wren, whole body	(1.1)[d]	—	3.5	—	3.5	—	—	—
quotient			3.2		3.2			

[a] Exposures predominantly to aquatic media and/or food sources.
[b] Mean of values reported for the site.
[c] Not determined because sample was not taken.
[d] Assumed same as value for mammals.
[e] Exposures predominantly to terrestrial media and/or food sources.

PCBs in aquatic biota. Whole body residues of 0.4 mg PCB/kg fresh weight are associated with reproductive toxicity in rainbow trout and hence have been proposed by EPA as a conservative limit for the protection of aquatic species (Eisler 1986). This level was exceeded in all the sites for the redbreast sunfish and stonerollers and was exceeded by greater than 5-fold at Sites 1, 2, and 3 (Table 6.75). The limit was also exceeded for crayfish at Site 1 (Table 6.75), but by factors of less than 2. Therefore, either crayfish receive a lower exposure to PCBs in EFPC surface water and sediment or they are better able to metabolize PCBs than fish.

PCBs in terrestrial biota. A value of 16 mg/kg body weight was chosen as the benchmark for toxicity to terrestrial biota (Eisler 1986). This benchmark was not exceeded in any terrestrial biota sample (Table 6.75).

Chlordane in aquatic biota. Nominal water concentrations of chlordane between 0.2 and 3.0 μg/L have been shown to cause adverse effects in aquatic organisms (Eisler 1990). Body burdens corresponding to these concentrations can be calculated using the published BCF for chlordane (Lyman et al. 1990) of 37,800:

$$\begin{aligned} C_{biota} &= C_{water} \times BCF \\ &= 0.2 \text{ to } 3\ \mu g/L \times 37{,}800\ L/kg \\ &= 76{,}000 \text{ to } 113{,}000\ \mu g/kg \end{aligned}$$

These calculated benchmark body burdens for chlordane in aquatic biota are much higher than the maximum of 100 μg/kg observed in EFPC biota (Sect. 6.2.3.2). Therefore, it is unlikely that chlordane in EFPC surface water poses a risk to aquatic biota.

Chlordane in terrestrial biota. A value of 300 μg/kg was chosen as the toxicological benchmark for chlordane in terrestrial biota (Eisler 1990). No EFPC terrestrial biota sample exceeded this benchmark (Table 6.76). Therefore, it is unlikely that chlordane body burdens pose a risk to biota in the EFPC floodplain.

PAHs in aquatic biota. PAHs are typically metabolized and excreted by fish and therefore do not usually accumulate to a high level in fish. They can, however, accumulate in benthic food chain organisms, which lack sufficient levels of metabolic enzymes to degrade PAHs (Black and Bauman 1990). Although there have been a number of studies on dose response of fish to PAHs in water, there is little evidence relating toxicity to body burden.

Toxicity values and BCFs for exposure of bluegill to fluorene were reported by Eisler (1987). Reported LC_{50} values for exposure of aquatic biota to PAHs ranged from ~300 μg/L to several thousand μg/L (Eisler 1987). In the studies reviewed by Eisler, fluorene at 500 μg/L

Table 6.75. Comparison of body burdens to criteria for toxicological effects from exposure to PCBs[a]

Organism, analysis	Criteria (mg/kg)	Sampling sites 1	2	3	4	5	6	Reference
Aquatic[a]								
Stoneroller, whole body[b]	0.4	8.1	7.4	2.0	0.8	1.1	—[c]	<0.01
quotient		**20.25**	**18.5**	5.0	2.0	2.8	—	<0.02
Redbreast sunfish, whole body	0.4	2.5	2.2	2.8	1.6	1.0	0.5	<0.02
quotient		6.25	5.5	7.0	4.0	2.5	1.25	<0.05
Crayfish, whole body	0.4	0.65	0.3	0.2	—	—	0.2	<0.02
quotient		1.6	0.7	0.4	—	—	0.4	<0.02
Heron, liver	16[d]	—	—	—	—	1.4	—	—
quotient		—	—	—	—	0.1	—	—
Terrestrial[e]								
Insect, whole body	16	0.3	0.35	0.34	0.14	0.11	0.02	0.02
quotient		0.02	0.02	0.02	0.008	0.007	0.001	0.004
Mouse, whole body	16	0.5	0.1	0.2	0.3	0.08	—	<0.02
quotient		0.03	0.006	0.01	0.02	0.005	—	<0.001
Shrew, whole body	16	—	—	—	1.4	1.0	—	—
quotient		—	—	—	0.09	0.06	—	—
Wren, whole body	16[d]	—	1.3	—	2.9	—	—	—
quotient		—	0.08	—	0.2	—	—	—

[a] Exposures predominantly to aquatic media and/or food sources.
[b] Mean of values reported for the site.
[c] Not determined because sample was not taken.
[d] Assumed same as value for mammals.
[e] Exposures predominantly to terrestrial media and/or food sources.

Table 6.76. Comparison of body burdens to criteria for toxicological effects from exposure to chlordane[a]

Organism, analysis	Criteria (µg/kg)	Sampling sites 1	2	3	4	5	6	Reference
Aquatic[a]								
Stoneroller, whole body[b]	750	105	110	58	50	63	—[c]	3
quotient		0.14	0.15	0.08	0.07	0.08	—	0.004
Redbreast sunfish, whole body	750	30	25	45	68	60	40	.2
quotient		0.04	0.03	0.06	0.09	0.08	0.05	0.003
Crayfish, whole body	750	10	BDL[d]	BDL	—	—	BDL	BDL
quotient		0.01			—	—		
Heron, liver	300[e]	—	—	—	—	12	—	—
quotient		—	—	—	—	0.04	—	—
Terrestrial[f]								
Insect, whole body	300	BDL	1.6	3.5	25	3	BDL	BDL
quotient			0.005	0.01	0.08	0.01		
Mouse, whole body	300	0.4	0.9	0.75	0.67	1.45	1	1
quotient		0.001	0.003	0.002	0.002	0.005	0.003	0.003
Shrew, whole body	300	—	—	—	BDL	8.8	—	—
quotient		—	—	—		0.03	—	—
Wren, whole body	300[e]	—	30	—	0.5	—	—	—
quotient		—	0.1	—	0.002	—	—	—

[a] Exposures predominantly to aquatic media and/or food sources.
[b] Mean of values reported for the site.
[c] Not determined because sample was not taken.
[d] Below sample detection limit
[e] Assumed same as value for mammals.
[f] Exposures predominantly to terrestrial media and/or food sources.

was toxic to 12% of the exposed fish, whereas 50% of fish were killed at 910 μg/L (the LC_{50}). BCFs ranged from 500 to 1800. Therefore, body burdens associated with 12% mortality would be

$$\begin{aligned} C_{biota} &= C_{water} \times BCF \\ &= 500\ \mu g/L \times 500 \text{ to } 1800\ L/kg \\ &= 250{,}000 \text{ to } 900{,}000\ \mu g/kg \end{aligned}$$

The maximum concentration of PAHs in fish collected from EFPC was approximately 50 μg/kg (Fig. 6.48), several orders of magnitude below values calculated above. Therefore, even allowing for a range of sensitivities among aquatic biota and of toxicities of various PAHs, it is unlikely that PAHs pose a risk to aquatic biota in EFPC.

PAHs in terrestrial biota. Bioaccumulation factors for terrestrial biota exposed to PAHs were not found. However, the published acute oral LD_{50} values of $\geq$ 50 mg/kg body weight (Eisler 1987) are several orders of magnitude above the apparent exposure concentrations in EFPC floodplain media. Therefore, it is unlikely that PAHs pose a risk to terrestrial biota in the EFPC floodplain.

Protection of piscivorous biota. Assessment endpoint 5c (Sect. 6.1) compares body burdens of contaminants in aquatic biota to concentrations expected to protect their predators from toxicological effects.

Mercury. A diet containing methylmercury at 1 mg/kg was lethal to mink after about 2 months (Eisler 1987), whereas a diet containing mercury at 0.05 to 0.10 mg/kg had only a minor effect on waterfowl (Eisler 1987). Therefore, a concentration of 0.1 mg/kg of total mercury in fish, crayfish, or aquatic insects was chosen as the criterion for this comparison. Detection limits for mercury in aquatic biota ranged from 0.15 to 0.22 mg/kg, so all samples could have exceeded this criterion even though mercury could not be detected. Table 6.77 shows the concentrations of mercury detected in aquatic biota and the quotients for protection of piscivorous biota. Quotients exceeding 10 are presented in bold type to emphasize the high values.

Table 6.77 shows that mercury concentrations in fish exceeded the criterion by tenfold or more at all sites except the farthest downstream from the Y-12 Plant (Site 6), while concentrations in crayfish exceeded 0.1 mg/kg by more than tenfold at the three uppermost sites (crayfish samples were not taken at Sites 4 and 5). Mercury concentrations in aquatic insects exceeded 0.1 mg/kg by at least tenfold at Sites 1 and 5. Therefore, piscivorous predators feeding

Table 6.77. Comparison of mercury body burdens with criterion for protection of piscivorous (0.1 mg/kg) and terrestrial predators (0.05 mg/kg)

Organism	Site 1	2	3	4	5	6	Reference
Aquatic[a]							
Stoneroller							
body burden[b]	3.1	6.4	1.9	0.7	0.7	0.4	<0.2
quotient	**31**	**64**	**19**	**7**	**7**	4	<2
Redbreast sunfish							
body burden	1.1	1.1	1.9	0.9	0.5	0.2	<0.2
quotient	**11**	**11**	**19**	**9**	**5**	2	<2
Crayfish							
body burden	6	3.3	0.7	—	—	0.2	<0.2
quotient	**60**	**33**	**7**	—	—	2	<2
Aquatic insect							
body burden	0.5	0.4	0.4	0.4	0.7	<0.13	<0.17
quotient	**10**	8	8	8	**14**	<2.6	<1.7
Heron							
liver	—	—	—	—	9.0	—	—
quotient	—	—	—	—	**180**	—	—
Terrestrial[c]							
Earthworm							
body burden	6.0	33.2	28.5	24.6	19.6	4.8	<0.3
quotient	**120**	**664**	**570**	**492**	**392**	**96**	<6
Terrestrial insect							
body burden	<0.14	0.2	3.2	<0.13	<0.12	<0.1	<0.09
quotient	<2.8	4	**64**	<2.6	<2.4	<2	<1.8
Mouse							
body burden	<0.3	0.9	<0.4	<0.2	<0.3	—	<0.4
quotient	<6	**18**	<8	<4	<6	—	<8
Shrew							
body burden	—	—	—	1.9	7.9	—	—
quotient	—	—	—	**38**	**158**	—	—
Wren							
body burden	—	3.5	—	3.5	—	—	—
quotient	—	**70**	—	**70**	—	—	—

[a] Exposures predominantly to aquatic media and/or food sources.
[b] Mean of values reported if multiple samples were taken (mg/kg).
[c] Exposures predominantly to terrestrial media and/or food sources.

exclusively in the creek above Site 5 would be at risk from dietary ingestion of mercury. The risk would be less with an expanded feeding range, or as the proportion of the diet coming from EFPC declines. Mean concentrations of mercury found in several fish species by TVA and BMAP (Tables 6.16 and 6.17) likewise exceeded 0.1 mg/kg.

PCBs. O'Connor and Pizza (1984) recommended PCB levels in fish diets of less than 0.5 mg/kg fresh weight based on their investigations with striped bass (Eisler 1986). Table 6.78 shows that the benchmark value of 0.4 mg PCBs was exceeded by Aroclor 1260 in stonerollers and redbreast sunfish at all sites, and in crayfish at Site 1. The exposure quotient was greater than 10 for stonerollers at Sites 1 and 2. Therefore, exposure to PCBs may present a risk to piscivorous aquatic biota, especially in the upper third of EFPC.

A limit of 100 μg PCB/kg diet was proposed to protect mink, which are unusually susceptible to PCBs (Eisler 1986). All samples of aquatic biota contained Aroclor 1260 at concentrations above 100 μg/mg, with exposure quotients ranging from a maximum of 81 for stonerollers at Site 1 to a minimum of 2 for crayfish at Site 6. Therefore, mink and other piscivorous biota with similar sensitivity to PCBs are at risk by ingestion of prey throughout the length of EFPC.

Chlordane. FWS (Eisler 1990) lists the guideline for the protection of predatory fish from chlordane in the diet at 100 μg/kg fresh weight. Table 6.79 shows that stonerollers at Sites 1 and 2 slightly exceeded the criterion for chlordane. None of the other aquatic biota samples had chlordane levels above 100 μg/kg. Therefore, chlordane appears to present a limited risk to piscivorous predators in EFPC.

PAHs. Information on toxicity of PAHs in the diet of piscivorous aquatic biota was not found. However, the high body burdens associated with potential toxicity (Sect. 6.4.1.1) imply that dietary exposure of aquatic biota to PAHs is not of concern.

Mallards fed a diet containing 4000 mg mixed PAHs/kg showed physiological effects but no visible signs of toxicity (Eisler 1987). The maximum concentrations of PAHs observed in aquatic biota sampled from EFPC were approximately 5 orders of magnitude below this value, providing adequate protection of ducks and, presumably, other piscivorous birds from PAH toxicity.

Protection of other predators. Assessment endpoint 7b covers other predators.

Mercury. A criterion of 0.05 mg mercury/kg in the diet has been proposed for protection of terrestrial mammals (Eisler 1987).

Table 6.78. Comparison of body burdens to criteria for protection of predators from exposure to chlordane[a]

Organism, analysis	Criteria (mg/kg)	Sampling sites 1	2	3	4	5	6	Reference
Aquatic[a]								
Stoneroller, whole body[b]	0.5	8.1	7.4	2.0	0.8	1.1	-[c]	<0.01
quotient		**16.2**	**14.8**	4.0	1.6	2.2	-	<0.02
Redbreast sunfish, whole body	0.5	2.5	2.2	2.8	1.6	1.0	0.5	<0.02
quotient		5.0	4.4	5.6	3.2	2.0	1.0	<0.04
Crayfish, whole body	0.5	0.65	0.3	0.2	—	—	0.2	<0.02
quotient		1.3	0.6	0.4	-	-	0.4	<0.04
Terrestrial[d]								
Insect, whole body	3	0.3	0.35	0.34	0.14	0.11	0.02	0.02
quotient		0.1	0.12	0.11	0.05	0.04	0.007	0.007
Mouse, whole body	3	0.5	0.1	0.2	0.3	0.08	—	<0.02
quotient		0.17	0.03	0.07	0.1	0.03	-	<0.007
Shrew, whole body	3	—	—	—	1.4	1.0	—	—
quotient		-	-	-	0.05	0.3	-	-
Wren, whole body	3	—	1.3	—	2.9	—	—	—
quotient		-	0.04	-	0.97	-	-	-
Heron, liver	3	—	—	—	—	1.4	-	—
quotient		-	-	-	-	0.5	-	-

[a] Exposures predominantly to aquatic media and/or food sources.
[b] Mean of values reported for the site.
[c] Not determined because sample was not taken.
[d] Exposures predominantly to terrestrial media and/or food sources.

Table 6.79. Comparison of body burdens to criteria for protection of predators from exposure to chlordane[a]

Organism, analysis	Criteria	Sampling sites						
	(µg/kg)	1	2	3	4	5	6	References
Aquatic[a]								
Stoneroller, whole body[b]	100	105	110	58	50	63	-[c]	3
quotient		1.05	1.1	0.6	0.5	0.6	-	0.03
Redbreast sunfish, whole body	100	30	23	45	60	60	40	2
quotient		0.3	0.2	0.4	0.6	0.6	0.4	0.02
Crayfish, whole body	100	10	BDL[d]	BDL	—	—	BDL	BDL
quotient		0.1			-	-		
Terrestrial[d]								
Insect, whole body	5000	BDL	1.6	3.5	25	3	BDL	BDL
quotient			3×10^{-4}	7×10^{-4}	5×10^{-3}	6×10^{-4}		
Mouse, whole body	5000	0.4	0.9	0.8	0.7	1.4	—	1
quotient		8×10^{-5}	2×10^{-4}	2×10^{-4}	1×10^{-4}	3×10^{-4}	-	3×10^{-3}
Shrew, whole body	5000	—	—	—	BDL	8.8	—	-
quotient		-	-	-		2×10^{-3}	-	
Wren, whole body	5000	—	30	—	0.5	—	—	-
quotient		-	6×10^{-3}	-	1×10^{-4}	-	-	
Heron, liver	5000	—	—	—	—	12	-	-
quotient		-	-	-	-	2×10^{-3}	-	

[a] Exposures predominantly to aquatic media and/or food sources.
[b] Mean of values reported for the site.
[c] Not determined because sample was not taken.
[d] Exposures predominantly to terrestrial media and/or food sources.

Earthworms contained high body burdens of mercury and other metals, most likely as a result of the contaminated soil in their digestive tracts. Earthworms at every sampling site exceeded criteria for dietary mercury; quotients ranged from nearly 100 to > 600 (Table 6.77). If that mercury were, in fact, bioavailable, and even if earthworms were only a small fraction of a predator's diet, exposure would be high. Terrestrial insect samples exceeded the criterion for mercury by a factor of 64 at Site 3. Otherwise, mercury was detected in terrestrial insects only at Site 2, with a quotient of 4. Assuming that predators of insects feed exclusively in the floodplain, it is likely that mercury in insects presents a high risk. Mice had detectable body burdens of mercury only at Site 2, with a quotient of 18, indicating some risk to predators at that site, but little risk elsewhere; however, mice predators have feeding ranges much larger than the floodplain.

Shrews had higher body burdens of mercury than mice, reflecting their greater consumption of insects and earthworms. Shrews had much higher body burdens of mercury than did terrestrial insects. However, they did not exhibit bioaccumulation of mercury above the total levels found in earthworms. Body burdens in wrens, which prey heavily on insects but not earthworms, were measured at Sites 2 and 4. The wrens had body burdens of ~3.5 mg/kg, exceeding the criterion for mercury by a factor of ~70. This may indicate a significant bioaccumulation of mercury from insects in the diet. Because the body burdens of earthworms and insects were not significantly lower at other sample sites, it is likely that if shrews and wrens are present at the other sites, then they would have body burdens similar to those observed. Therefore, it appears that both shrews and wrens in the EFPC floodplain are at risk. Sect. 4 provides ARARs that will be further used in the quotient and similar approaches during the FS-EIS.

PCBs. A diet containing 3 mg PCB/kg fresh weight has been shown to result in high PCB concentration in the eggs of screech owls (McLane and Hughes 1984). Whole-body values for toxicity of PCBs in terrestrial mammals were not found, so the benchmark for birds was used to evaluate potential toxicity to terrestrial mammals as well. Table 6.78 compares Aroclor 1260 concentrations in terrestrial biota samples to the benchmark value of 3 mg/kg. None of the EFPC terrestrial biota samples exceeded the benchmark value.

Chlordane. The proposed benchmark for protection of terrestrial biota from chlordane in the diet is 5 mg/kg (Eisler 1990). Table 6.79 shows that chlordane body burdens in terrestrial biota sampled from the EFPC floodplain were several orders of magnitude below this level. Therefore, chlordane does not propose a risk to predators of terrestrial biota in the EFPC floodplain.

PAHs. Although experimental studies of carcinogenesis by PAHs are abundant, there is little information on direct toxicity of PAHs to terrestrial biota. Acute oral LD_{50} values of 50 mg/kg body weight for benzo(a)pyrene and 700 mg/kg body weight for phenanthrene were reported by Eisler (1987). If food is consumed at a rate of 20% of body weight daily and an arbitrary safety factor of 100 is applied to account for chronic exposure, the corresponding limits on concentration in food would be 2.5 mg benzo(a)pyrene/kg diet and 35 mg phenanthrene/kg diet. These values are several orders of magnitude above contaminant levels observed in any EFPC biota samples. Therefore, it is concluded that PAHs do not present a risk to terrestrial predators in the EFPC system.

Effect of diet and feeding range on exposure of predators. In the preceding discussion, calculations of exposure quotients for predators assumed that each kind of prey (fish, earthworms, mice, shrews, wrens) was the sole or predominant component of the predators' diets. However, few predators restrict their diet to one kind of prey. Thus, shrews may accumulate soil contaminants from insects, other arthropods, worms, or any other available small animals. Similarly, shrews should not be considered the exclusive prey of such predators as owls, hawks, and foxes. Thus, predators receive exposures from varying amounts of contaminants in different prey. To provide an organized way of dealing with these multiple exposures, several assumptions are made about dietary exposure:

- The food source for predators feeding in the EFPC floodplain is considered to be a mix of prey including insects, earthworms, small mammals, and birds (represented by wrens).

- Body burden data for each prey taxon are aggregated by averaging contaminant body burdens for the six EFPC ecological sampling sites.

- Aggregate body burdens will be associated spatially with the band of contamination bordering much of the creek and in the zones of sediment deposition that have resulted in more extensive contamination (for example, at Sites 2 and 3). Prey taken outside these areas of contamination are assumed not to have contaminant body burdens.

- Composite exposure concentrations to predators are calculated by multiplying the assumed fraction of diet composed by each biota type by its aggregate body burden.

The feeding range of the predators must also be considered in this analysis. The smaller the feeding range, the more likely it is that a predator may feed entirely within the contaminated area

of the floodplain. Conversely, the larger the feeding range, the less will be the influence of contaminated prey on the body burden of the predator.

For much of the creek's length, elevated mercury levels were found only in the soil sample adjacent to the creek edge. The feeding ranges of large predators such as hawks, owls, and foxes are large [e.g., 25 to 250 ha (62 to 620 acres)]. Therefore, they are feeding both inside and, primarily, outside the contaminated areas of the EFPC floodplain. The feeding ranges of shrews or insectivorous birds, which can be less than 1 ha (2.5 acres), may be limited to contaminated areas, especially in areas where contaminated sediments have been deposited at some distance from the creek.

The following discussion presents an approach to quantifying the contribution of prey from contaminated areas to the diet of a typical predator at EFPC. Although the shape of the actual territorial units may not be regular because of geography or habitat, for this approach they will be assumed to be so to facilitate analysis. The adjoining ranges of predators are assumed to be more or less exclusive territories that can be conceived of as a series of adjacent regular hexagons. To ensure that exposures are modeled conservatively, the hexagons (feeding ranges) can be arranged so that they are centered on the creek, putting the largest possible contaminated area within each range. By geometric relationships, the size of the rectangle representing the contaminated area can be calculated as a fraction of the entire range. The calculated fractions of a hypothetical predator's range is made up by contaminated zones of different sizes. An owl with a feeding range of 100 ha (250 acres) would typically obtain about 4.25% of its diet from the contaminated area. To obtain more than half its diet from the contaminated zone, a predator's range would have to be less than 0.8 ha (2 acres).

Dietary exposures for predators were modeled by considering the size of the contaminated zone, feeding range, dietary content, and body burdens in prey. Exposures were calculated using the following data and assumptions:

- The contaminated zone extends no more than about 20 m (65.6 ft) on each side of the creek. This is true for much of the length of the floodplain (Sect. 3).

- Typical feeding ranges taken from published sources and the calculated fraction of range within the contaminated zone of the floodplain are shown in Table 6.80.

Table 6.80. Risk factors for exposure of terrestrial predators

Predator	Reported range (ha)	Fraction of range within contaminated area	Assumed dietary contribution of prey taxa				
			Bird (Wren)	Shrew	Mouse	Insect	Earthworm
Hawk	250[a]	0.03	0.3	0.04	0.36	0.3	0
Owl	100[b]	0.04	0.3	0.04	0.36	0.3	0
Fox	25[c]	0.09	0.05	0.09	0.81	0.05	0
Wren	0.8[d]	0.5	0	0	0	1	0
Shrew	0.3[e]	0.8	0	0	0	0.5	0.5

[a] Austing and Terres 1964
[b] Johnsquard 1988
[c] Chapman and Feldhamer 1982
[d] Peterson and Peterson 1980
[e] Burt and Grossenheider 1976

- The contents of typical diets of several potential EFPC floodplain predators are estimated to be as in Table 6.80. Wrens represent all insectivorous birds. Prey that receive their dietary exposures mainly from plants (e.g., seed-eating birds and rabbits) are represented by mice. Based on capture frequencies (~4.5% of all small mammals captured), shrews were assumed to comprise 10% of the assumed fraction of small mammals in the diet.

- The mean concentrations of mercury and PCBs in prey taxa were calculated from measured data presented in Sect. 6.2. It was assumed that methyl-mercury is a small fraction of total mercury in all terrestrial biota, whereas all mercury in aquatic insects was assumed to be methyl-mercury. The fractions of aquatic and terrestrial insects in the diet were calculated from the recovery of insects during ecological sampling (67.5 g aquatic insects, 266 g terrestrial insects), and the exposure concentrations were weight-averaged using these data.

The calculated results are shown in Table 6.81. These results show that shrews and wrens may receive a dietary intake of mercury substantially above the benchmark value of 0.05 mg/kg diet, whereas hawks, owls, and foxes may slightly exceed the benchmarks for dietary exposure. Dietary exposure to methyl-mercury was below the benchmark of 0.05 mg/kg diet for birds and a conservative value of 0.5 mg/kg diet for mammals (Eisler 1987). For PCBs, the calculations show that hawks, owls, and foxes should receive exposures less than the benchmark level of 0.5 mg/kg diet; body burden data for earthworms and insects, the primary diet of shrews and wrens, are not available for PCBs.

The results support the conclusion that predators feeding mainly on worms and insects are at risk from ingestion of mercury. The risk to top predators feeding on birds and small mammals is smaller but may be significant.

Potential impacts of loss of prey. Terrestrial predators may be impacted by loss of prey due to contaminant effects or physical stressors. If it is assumed that predators and prey populations are in equilibrium, any reduction in abundance of suitable prey will force predators to expand their hunting range, change their diet, survive on less food than optimal, or suffer diminished reproductive capacity or death as a result of lack of food. The observed densities of terrestrial populations in the EFPC floodplain were highly variable (Tables 6.57, 6.58, 6.65, and 6.66), but they generally increased with distance downstream, probably reflecting both a decrease in exposure to contaminants and changes in habitat. The mean densities of various terrestrial taxa averaged over the six ecological sampling sites in the EFPC floodplain were compared to their

Table 6.81. Calculated dietary exposures of terrestrial predators

Predator	Calculated concentration (mg/kg)			Exposure quotient		
	Inorganic mercury	Methyl-mercury	Aroclor 1260	Inorganic mercury[a]	Methyl-mercury[b]	Aroclor 1260[c]
Hawk	0.039	4.6×10^{-4}	0.021	0.78	0.01	0.04
Owl	0.062	7.2×10^{-4}	0.033	1.2	0.14	0.07
Fox	0.068	2.4×10^{-4}	0.034	1.4	0.0002	0.07
Wren	2.04	0.022	—[a]	41	0.44	—
Shrew	9.58	0.027	—	192	0.027	—

[a] Benchmark = 0.05 mg/kg for birds, 1.1 mg/kg for mammals
[b] Benchmark = 0.05 for birds, 0.5 mg/kg for mammals
[c] Benchmark = 0.5 mg/kg

densities at the respective reference areas to demonstrate potential population impacts of the EFPC floodplain (Table 6.82). The results of this comparison showed:

- Small mammals were trapped more frequently in the EFPC floodplain by a factor of 3.3.
- Earthworms were observed to be more abundant by a factor of 2.9.
- Flying insects with aquatic juvenile stages were more abundant by a factor of 1.9.
- Flying insects with terrestrial juvenile stages were less abundant in the EFPC floodplain by a factor of 0.4.
- All insects combined were 85% as abundant in the EFPC floodplain as at the reference site.

Therefore, in comparison to the reference sites, the EFPC floodplain has abundant earthworms and small mammals, implying that their predators should not be impacted by lack of food. However, insectivorous predators whose feeding range is restricted to the EFPC floodplain may be impacted by a reduction of numbers of insects, especially those with terrestrial juvenile stages. If the overall abundance of insectivorous predators is correlated with the abundance of insect prey, and if the abundance of insects observed during the ecological survey is representative of the EFPC floodplain, the potential impact on the insectivorous predators should be less than the 20% reduction in survival recommended by Suter (1993) as a benchmark for significant ecological effects. Therefore, it is unlikely that reductions in prey populations by either contaminants or physical stressors in the EFPC floodplain have had a major impact on terrestrial predator populations.

6.4.1.2 Weight-of-evidence analysis

The following weight-of-evidence analysis considers several propositions. These propositions are conclusions based on the results of the ERA field sampling, as well as the historical studies on EFPC. Various data (including several assessment endpoints discussed in Sect. 6.1.3.1, as well as measurement endpoints) on community structure and/or contaminant concentrations in physical media and biota were incorporated into each proposition, as appropriate, and were evaluated against each of five criteria during the weight-of-evidence analysis.

Table 6.82. Relative abundance of prey taxa in the EFPC floodplain

Prey taxon	Abundance measure	Ecological risk			Source of data
		Mean EFPC floodplain	Reference	Ratio	
Small mammals	Total captures per effort	9.96	2.96	3.36	Table 6.49
Earthworms	Individuals per m^2	118.5	40	2.96	Table 6.58
Flying insects, aquatic juveniles	Mean individuals per sample	1022	548	1.86	Table 6.55
Flying insects, terrestrial juveniles	Mean individuals per sample	529	1248	0.41	Table 6.56
Flying insects, total	Mean individuals per sample	776	898	0.86	Table 6.55 and 6.56

This section begins with a list of the major observations from the ERA field sampling, followed by a list of the major observations from historical studies on EFPC. Next, the propositions are presented, followed by the weight-of-evidence analysis. The emphasis is on aquatic resources. The weight-of-evidence for terrestrial resources is provided following the one for aquatic resources.

Major observations from the ERA aquatic field sampling

The following measurement parameters generally were highest closest to the Y-12 Plant (Site 1), then decreased downstream (to Sites 3 or 4), and were usually greater at all sites in EFPC compared to the reference site:

- total mercury concentrations in surface water and sediments (Fig. 6.43);
- whole-body burdens of mercury in fish, benthic macroinvertebrates, and crayfish (except that maximum mercury body burdens in stonerollers and redbreast sunfish occurred at Sites 2 and 3, respectively, and the body burdens in stonerollers from the three most downstream sites in EFPC exceeded the levels in stonerollers from the reference site) (Figs. 6.42, 6.43, 6.49);
- Aroclor 1260 whole body burdens in fish, benthic macroinvertebrates, and crayfish (except maximum body burdens in redbreast occurred at Site 3) (Figs. 6.44, 6.50);
- chlordane, dieldrin, and heptachlor body burdens in stonerollers (Fig. 6.46);
- PAHs [benzo(a)anthracene, benzo(a)pyrene, benzo(g,h,i)perylene, chrysene, indeno(1,2,3-c,d)pyrene] body burdens in stonerollers (Fig. 6.47);
- uranium (Fig. 6.48), arsenic, chromium, and zinc body burdens in stonerollers;
- chlordane, dieldrin, and DDE body burdens in crayfish (Fig. 6.51); and
- proportion of fish species classified as tolerant of degraded water quality conditions (including toxic chemical exposures) (Figs. 6.58 and 6.59; species names and their relative abundance are listed in Table 6.51).

The following measurement parameters generally were lowest closest to the Y-12 Plant, then increased downstream, and were usually lower at all sites in EFPC compared to the reference site:

- EPT richness (Fig. 6.64),
- taxonomic diversity indices of fish (species) and benthic macroinvertebrates (families) (Fig. 6.73),
- taxonomic richness of fish and benthic macroinvertebrates (Fig. 6.71).

Major observations from historical aquatic studies on EFPC

The following measurement parameters generally were highest closest to the Y-12 Plant (EFK 23.4 to EFK 22.2), then decreased downstream, and were usually greater at all sites in EFPC compared to the reference site:

- mercury concentration in fish fillets (Van Winkle et al. 1984; TVA 1985; Loar 1992; Hinzman 1992) (Tables 6.16, 6.17, 6.19);
- total PCBs in sunfish fillets (Loar 1992; Hinzman 1992) (Tables 6.18 and 6.20);
- percentage of fish species classified as tolerant of degraded conditions (adapted from Loar 1992; Hinzman 1992);
- mercury, cadmium, copper, lead, nickel, silver, and zinc concentrations in periphyton samples collected from rocks instream (Hinzman 1992) (Table 6.23); and
- mortality of caged fingernail clams (Kornegay 1992; Hinzman 1992).

The following measurement parameters generally were lowest closest to the Y-12 Plant, then increased downstream (maximum usually at EFK 13.8 or EFK 10.6), and were usually lower at all sites in EFPC compared to the reference site:

- taxonomic richness of fish and benthic macroinvertebrates (Hinzman 1992),
- EPT richness (Hinzman 1992),

- benthic macroinvertebrate diversity (Hinzman 1992), and

- natality of fingernail clams (Kornegay 1992).

Historical studies conducted during March 1986 - October 1988 by BMAP on the ambient toxicity of EFPC surface water (7-d tests using *Ceriodaphnia*, fathead minnows, and surface water samples from 6 sites downstream from the former New Hope Pond) have provided some evidence of reduced growth of fathead minnows at sites closest downstream from New Hope Pond (Loar 1992). However, most of the tests generally concluded that there was no consistent pattern or evidence of toxicological impacts to the test species at the sites in EFPC (Hinzman 1992). Complete results of the ambient toxicity tests that were conducted from 1989 to the present were not available as of the writing of this ERA report.

Propositions

The following three propositions describe impacts on aquatic biota in EFPC that appear to be attributable to ongoing contaminant releases from the Y-12 Plant. The three propositions each begin with the statement, "Continual release of water-borne contaminants (including dissolved and particle-bound) such as mercury, uranium, other inorganics, pesticides, PAHs, and Aroclor 1260 from the Y-12 Plant," and are as follows:

(1) Continual release of water-borne contaminants . . . from the Y-12 Plant are the largest source of the elevated whole body burdens of these contaminants in fish, benthic macroinvertebrates, and crayfish in EFPC, which results in risk from toxicological effects to the indicator organisms and risks to piscivorous predators feeding on the organisms.

(2) Continual release of water-borne contaminants . . . from the Y-12 Plant impact fish community structure in EFPC, resulting in communities dominated by species tolerant of degraded water quality conditions.

(3) Continual release of water-borne contaminants . . . from the Y-12 Plant result in reduced taxonomic richness and diversity of the fish and benthic macroinvertebrate communities in EFPC compared to the same measurements in communities in the reference site.

Support for the propositions is presented in the following subsections.

Analysis

Temporal associations. No data were available on the body burdens of mercury, Aroclor 1260, or any of the other COCs in fish or benthic macroinvertebrates collected from EFPC prior to DOE operations. However, historical investigations have revealed that mercury and PCBs have been continually released from the Y-12 Plant for many years, and ongoing monitoring studies indicate that these contaminants continue to be released into EFPC and bioaccumulate in fish (Loar 1992; Hinzman 1992). In addition, mercury and PCB contaminant concentrations in sunfish from EFPC have increased in response to sediment-disturbing construction activities at the Y-12 Plant and decreased after completion of remedial action activities at the plant, which reduced the concentration of mercury discharged into EFPC (Hinzman 1992). Although the historical data contain little information on other contaminant concentrations in aquatic biota in EFPC, the data for mercury and PCBs indicate a clear temporal association between the release of these two contaminants from the Y-12 Plant and their subsequent bioaccumulation in aquatic biota downstream in EFPC.

Historical studies have not clearly stated what, if any, temporal relationships exist between contaminant concentrations released from the Y-12 Plant and subsequent effects on the percentage of fish species classified as tolerants (Proposition 2) or fish and benthic macroinvertebrate taxonomic richness and diversity (Proposition 3). Nonetheless, studies since 1985 (Loar 1992; Hinzman 1992) have shown that several variables (such as the percentage of captured fish species that are classified as tolerant of degraded water quality conditions, fish and benthic macroinvertebrate taxa richness, and species diversity) have consistently indicated that aquatic biota in EFPC are adversely impacted in comparison to biota in a reference stream. Some aspects of the aquatic community are recovering, but these aspects have not recovered per the BMAP study.

Spatial associations. Two types of information were used to evaluate spatial associations. The first type of information was the longitudinal trend in the measurement parameters within EFPC, indicating the influence of a major point source of contaminants. A strong longitudinal trend was indicated if: (1) the magnitude of the measurement parameters consecutively increased or decreased at the first three sampling sites downstream from the Y-12 Plant, beginning at Site 1 or (2) the magnitude of the measurement parameters increased or decreased in a consistent trend among at least two out of three of the sites in EFPC. The second information used to determine spatial associations was the qualitative comparison of measurement parameters in EFPC against the same parameters in the reference stream. A strong spatial association of effect with

EFPC contaminants was indicated when the magnitude of the measurement parameters from at least two out of three of the sites in EFPC exceeded the values observed at the reference site.

Spatial associations between the release of contaminants from the Y-12 Plant and subsequent bioaccumulation in aquatic biota (Proposition 1) were determined for contaminants whose concentrations were above the detection limit in biota (Table 6.83). Body burdens of numerous contaminants in stonerollers, redbreast sunfish, and crayfish exhibited strong spatial association with the Y-12 Plant (Table 6.84). Mercury and Aroclor 1260 body burdens in all of the aquatic biota except benthic macroinvertebrates displayed strong spatial associations with respect to longitudinal trends as well as the percentage of body burdens in EFPC that exceeded those from the reference site. Spatial associations could not be determined for Aroclor 1260 in benthic macroinvertebrates because only two samples were obtained with sufficient quantity for PCB and pesticide analysis.

In stonerollers, body burdens of 14 contaminants displayed strong spatial associations in EFPC, which were 4 metals, Aroclor 1260, 5 PAHs, and 4 pesticides (Table 6.84). Of these, 9 showed strong longitudinal trends, whereas 12 were larger in magnitude at EFPC compared to the reference site. Seven of the contaminants displayed both trends.

In contrast, body burdens of only 6 contaminants in redbreast sunfish showed strong spatial associations in EFPC (Table 6.84). The 6 contaminants were mercury, zinc, Aroclor 1260, and 3 pesticides. Of these contaminants, only 3 showed strong longitudinal trends, while 5 were of larger magnitude in EFPC than at the reference site. Only 2 of the 6 contaminant body burdens displayed strong longitudinal trends and larger magnitude in relation to the reference site.

Crayfish in EFPC had body burdens of 7 contaminants that showed strong spatial associations, mercury, zinc, Aroclor 1260, 2 PAHs, and 2 pesticides (Table 6.84). Of these, 5 had strong longitudinal trends, and 6 were larger in magnitude in comparison to the reference site. Four of the contaminant body burdens displayed both trends.

In benthic macroinvertebrates, only the body burdens of mercury and uranium had strong spatial associations in EFPC (Table 6.84). Both contaminants were larger in magnitude in EFPC in comparison to the reference site. Strong longitudinal trends were not observed for body burdens of either contaminant.

Stonerollers had twice as many contaminant body burdens with strong spatial associations in comparison to redbreast sunfish or crayfish. The reason why stonerollers contained so many contaminants that displayed strong spatial associations is probably due to the combination of: (1)

Table 6.83. Body burden spatial association evaluation for longitudinal trends and larger magnitudes in relation to the reference site

Contaminant in Biota	Number of EFPC sites with clear longitudinal trend considering Sites 1 to 3	Number of EFPC sites with clear longitudinal trend considering Sites 1 to 6	Number of EFPC sites that exceed the Reference value
Aroclor 1260 in Stoneroller	3 of 3 (100%)	4 of 5 (80%)	5 of 5 (100%)
Aroclor 1260 in Redbreast	(max at Site 3) 2 of 3 (67%)	5 of 6 (83%)	6 of 6 (100%)
Aroclor 1260 in Crayfish	3 of 3 (100%)	3 of 4 (75%)	4 of 4 (100%)
Aroclor 1260 in Benthic Macroinvertebrates	2 of 2 (100%)	2 of 2 (100%)	NA
Benzo(g,h,i)perylene in Stoneroller	(max at Site 1) 2 of 3 (67%)	3 of 5 (60%)	5 of 5 (100%)
Benzo(g,h,i)perylene in Benthic Macroinvertebrates	2 of 2 ↑ (100%)	2 of 2 ↑ (100%)	NA
Indeno(1,2,3-cd)pyrene in Stoneroller	3 of 3 (100%)	5 of 5 (100%)	5 of 5 (100%)
Indeno(1,2,3-cd)pyrene in Benthic Macroinvertebrates	2 of 2 ↑ (100%)	2 of 2 ↑ (100%)	NA
Chrysene in Stoneroller	3 of 3 (100%)	4 of 5 (80%)	3 of 5 (60%)
Chrysene in Crayfish	(max at Site 3) 2 of 3 (67%)	(max at Site 3) 2 of 3 (67%)	2 of 3 (67%)
Chrysene in Benthic Macroinvertebrates	2 of 2 (100%)	2 of 2 (100%)	NA
Benzo(a)pyrene in Stoneroller	3 of 3 (100%)	4 of 5 (80%)	5 of 5 (100%)
Benzo(a)pyrene in Crayfish	3 of 3 ↑ (100%)	3 of 3 ↑ (100%)	3 of 3 (100%)
Benzo(a)pyrene in Benthic Macroinvertebrates	2 of 2 ↑ (100%)	2 of 2 ↑ (100%)	NA

Table 6.83. (continued)

Contaminant in Biota	Number of EFPC sites with clear longitudinal trend considering Sites 1 to 3	Number of EFPC sites with clear longitudinal trend considering Sites 1 to 6	Number of EFPC sites that exceed the Reference value
Alpha Chlordane in Stoneroller	(max at Site 2) 2 of 3 (67%)	3 of 5 (60%)	5 of 5 (100%)
Alpha Chlordane in Redbreast	[ambiguous trend] max at Site 3	[2 trends] Sites 1 to 4: 3 of 4 (75%)↑; Sites 4 to 6: 3 of 3 (100%)	6 of 6 (100%)
Alpha Chlordane in Crayfish	2 of 2 (100%)	2 of 2 (100%)	2 of 2 (100%)
Benzo(a)anthracene in Stoneroller	(max at Site 1) 2 of 3 (67%)	3 of 5 (60%)	4 of 5 (80%)
Benzo(a)anthracene in Crayfish	3 of 3 ↑ (100%)	3 of 3 ↑ (100%)	3 of 3 (100%)
Dieldrin in Stoneroller	3 of 3 (100%)	4 of 5 (80%)	5 of 5 (100%)
Dieldrin in Redbreast	(max at Site 3) 2 of 3 (67%)	4 of 6 ↑ (67%)	6 of 6 (100%)
Dieldrin in Crayfish	3 of 3 (100%)	3 of 3 (100%)	NA
DDE in Stoneroller	3 of 3 (100%)	4 of 5 (80%)	4 of 4 (100%)
DDE in Redbreast	(max at Site 2) 2 of 3 (67%)	2 of 3 (67%)	3 of 3 (100%)
DDE in Crayfish	2 of 3 (67%)	2 of 3 (67%)	3 of 3 (100%)
Mercury in Stoneroller	(max at Site 2) 2 of 3 (67%)	4 of 6 (67%)	6 of 6 (100%)

Table 6.83. (continued)

Contaminant in Biota	Number of EFPC sites with clear longitudinal trend considering Sites 1 to 3	Number of EFPC sites with clear longitudinal trend considering Sites 1 to 6	Number of EFPC sites that exceed the Reference value
Mercury in Redbreast	3 of 3 ↑ (100%)	4 of 6 (Sites 3 to 6) 67%)	6 of 6 (100%)
Mercury in Crayfish	3 of 3 (100%)	4 of 4 (100%)	4 of 4 (100%)
Mercury in Benthic Macroinvertebrates	[ambiguous trend] max at Site 2	[ambiguous trend] max at Site 2	4 of 4 (100%)
Arsenic in Stoneroller	(max at Site 2) 2 of 3 (67%)	2 of 3 (67%)	3 of 3 (100%)
Chromium in Stoneroller	(max at Site 2) 2 of 3 (67%)	(max at Site 2) 5 of 6 (83%)	2 of 6 (33%)
Chromium in Redbreast	2 of 3 ↑ (67%)	[ambiguous trend] (max at Site 2)	0 of 6 (0%)
Chromium in Benthic Macroinvertebrates	2 of 3 ↑ (67%)	3 of 4 ↑ (75%)	2 of 4 (50%)
Zinc in Stoneroller	3 of 3 (100%)	6 of 6 (100%)	2 of 6 (33%)
Zinc in Redbreast	(max at Site 1) 2 of 3 ↑ (67%)	5 of 6 (83%)	4 of 6 (67%)
Zinc in Crayfish	(max at Site 1) 2 of 3 (67%)	3 of 4 (75%)	4 of 4 (100%)
Zinc in Benthic Macroinvertebrates	(max at Site 2) 1 of 3 (33%)	1 of 4 (25%)	2 of 4 (50%)

Table 6.83. (continued)

Contaminant in Biota	Number of EFPC sites with clear longitudinal trend considering Sites 1 to 3	Number of EFPC sites with clear longitudinal trend considering Sites 1 to 6	Number of EFPC sites that exceed the Reference value
Heptachlor in Stoneroller	3 of 3 (100%)	3 of 5 (60%)	5 of 5 (100%)
Heptachlor in Redbreast	(max at Site 3) 2 of 3 ↑ (67%)	(max at Site 5) 4 of 6 ↑ (67%)	5 of 6 (83%)
Uranium in Stoneroller	3 of 3 (100%)	4 of 4 (100%)	4 of 4 (100%)
Uranium in Benthic Macroinvertebrates	(max at Site 2) 2 of 3 ↑ (67%)	(max at Site 2) 3 of 4 ↑ (75)	4 of 4 (100%)

NA = insufficient sample collected to perform analysis or sample not collected
↑ = increasing trend downstream from the Y-12 Plant.

Table 6.84. Summary of the strong spatial associations based on longitudinal trends or the layer magnitude of the measurement parameters in EFPC in comparison to the reference site.

Measurement parameter		Strong longitudinal trend	Magnitude of measurement parameter greater than the reference site in ≥ 75% of the sites in EFPC
Whole Body Contaminant Levels			
Stonerollers	- arsenic	-	Y
	- mercury	-	Y
	- uranium	Y	Y
	- zinc	Y	-
	- Aroclor 1260	Y	Y
	- alpha chlordane	-	Y
	- benzo(a)anthracene	-	Y
	- benzo(a)pyrene	Y	Y
	- benzo(g,h,i)perylene	-	Y
	- chrysene	Y	-
	- DDE	Y	Y
	- Dieldrin	Y	Y
	- Heptachlor	Y	Y
	- indeno(1,2,3-cd)pyrene	Y	Y
Redbreast			
	- mercury	Y	Y
	- zinc	Y	-
	- Aroclor 1260	Y	Y
	- alpha chlordane	-	Y
	- DDE	-	Y
	- Dieldrin	-	Y
Crayfish			
	- mercury	Y	Y
	- zinc	-	Y
	- Aroclor 1260	Y	Y
	- benzo(a)anthracene	Y[a]	Y
	- benzo(a)pyrene	Y	Y
	- DDE	-	Y
	- Dieldrin	Y	-
Benthic Macroinvertebrates			
	- mercury	-	Y
	- uranium	-	Y

Table 6.84. (continued)

Measurement parameter	Strong longitudinal trend	Magnitude of measurement parameter greater than the reference site in ≥ 75% of the sites in EFPC
Fish Population / Community Parameters		
Percent of species classified as Tolerant	Y[b]	-
Total Richness	Y	-
Species Diversity (H')	Y	Y
Benthic Microinvertebrates Population / Community Parameters		
Total Richness	-	Y
EPT Richness	-	Y
Family Diversity (H')	-	Y

[a] increasing
[b] Y = yes

water-borne contaminants released from the Y-12 Plant, (2) the stoneroller's diet, which consists of periphyton, and (3) possible toxicokinetic factors such as higher dietary uptake efficiencies. Thus, water-borne contaminants from the Y-12 Plant would tend to concentrate on and in periphyton, through adsorption onto algal and diatom cells, as well as bioconcentration of the contaminants into these cells. Corroborative evidence for this can be found in the Y-12 Plant BMAP investigations (Hinzman 1992), which reported that the concentrations of metals (including mercury) in periphyton were highest at sites closest to the Y-12 Plant and decreased downstream (Table 6.23). Periphyton would provide stonerollers with an exposure dose of the contaminants that were concentrated within its matrix. Redbreast sunfish do not feed on periphyton, so they would not receive the dietary exposure of concentrated contaminants that the stonerollers receive. Crayfish are scavengers, so periphyton would not comprise a major portion of their diet. Although some benthic macroinvertebrates do feed on periphyton, those specific taxa were not analyzed separately from the composite benthic macroinvertebrate samples.

Among the 4 taxa of aquatic biota that were sampled for contaminant body burdens, 14 different contaminants displayed strong spatial associations, due either to strong longitudinal trends, or to larger magnitudes in comparison to the reference site, or both (Table 6.84). Thus, the data clearly indicate numerous strong spatial associations in support of Proposition 1.

The percentage of captured fish species classified as tolerants at each site (Proposition 2) was greatest closest to the Y-12 Plant, then steadily decreased downstream to Site 6. However, the percentage of tolerant species in only 4 of 6 sites in EFPC exceeded the percentage at the reference site. Due to the strong longitudinal trend of decreasing percentages of tolerant fish species downstream from the Y-12 Plant, the spatial association is strong for Proposition 2.

Longitudinal trends in fish and benthic macroinvertebrate taxonomic richness and diversity (Proposition 3) in EFPC were inconsistent. For example, fish species richness increased downstream from Site 2 to Site 5, but the species richness at Site 5 was the same as at the site closest and furthest from the Y-12 Plant (Sites 1 and 6, respectively). Fish species diversity increased downstream from the Y-12 Plant, except for a decrease at Site 3. Taxonomic richness of benthic macroinvertebrates increased at 2 of the 4 sites between Sites 1 and 4, and reached a maximum at Site 4, more than 10 km (6 mi) downstream from the Y-12 Plant. Benthic macroinvertebrate diversity steadily increased downstream between Sites 1 to 4, then progressively decreased at the last two sites in EFPC. Thus, based on longitudinal trends, the spatial associations for fish and benthic macroinvertebrate diversity were strong, whereas spatial associations for taxonomic richness were weak.

It is noteworthy that although taxonomic richness (and presumably diversity) might be expected to increase in a typical stream as its drainage area or stream order increase (Fausch et al. 1984), the argument may be less applicable to EFPC because it does not possess the characteristics of a true headwater stream due to the augmented flow from the Y-12 Plant. Therefore, the increased taxonomic richness and diversity downstream from the Y-12 Plant is probably due more to the reduction in toxicant concentrations downstream than to the increase in drainage area.

Spatial associations based on the magnitudes of fish and benthic macroinvertebrate taxonomic richness and diversity indices in EFPC, compared to the magnitudes at the reference site, were all strong (Table 6.84). Taxonomic richness and diversity were lower at every site in EFPC compared to the reference site, except for fish species diversity at the two most downstream sites in EFPC. Thus, the data indicate a strong spatial association between effect and location in support of Proposition 3.

Two additional facts strengthen the arguments for spatial association. First, water-borne contaminants that are released from Lake Reality (the Y-12 Plant) discharge immediately and directly into EFPC. Secondly, fish and benthic macroinvertebrates are restricted to their aquatic habitat, so their exposure to water-borne contaminants is virtually assured. These two facts, coupled with the observations supporting the three propositions, clearly demonstrate a strong spatial association.

Stressor-response. Stressor-response associations are most clearly demonstrated when there are strong correlations between the COC exposure concentrations and the magnitude of some response variable in biota (for example, the percentage of captured fish species that are classified as tolerants). In this study, establishing clear stressor-response associations for COCs was difficult for three reasons. First, except for mercury and zinc, most of the COCs in surface water and many in sediments were below detection limits; actual concentrations were, therefore, unknown. Second, the measured concentration of COCs in surface waters represent a single moment in time and may not accurately reflect the concentration to which the biota were exposed over a long-term duration. Third, the numerous COCs increase the potential for antagonistic or synergistic effects between COCs, and the magnitude of these effects is unknown. Therefore, for this weight-of-evidence analysis, stressor-response associations were evaluated by one of three methods, depending on whether the aqueous concentrations of the COCs were above detection limits.

If the COC aqueous concentrations were above detection limits, then linear correlation coefficients were determined between those concentrations and the magnitude of the response variables of interest (i.e., body burdens for Proposition 1, percentage of tolerant fish species for Proposition 2, and fish and benthic macroinvertebrate taxonomic richness and diversity for Proposition 3). Statistically significant correlations indicated a strong stressor-response. If the COC aqueous concentrations were below detection limits, the stressor-response association was determined by other methods, depending on the proposition. For Proposition 1, stressor-response association was indicated if the magnitude of the body burdens of COCs in the aquatic biota decreased downstream from the Y-12 Plant. The rationale for using this evaluation procedure is as follows: In the absence of aqueous contaminant concentrations in EFPC, the body burden concentrations of those contaminants in aquatic biota would provide evidence of exposure to the contaminant (exposure to the stressor). Although body burdens of contaminants in aquatic biota can be influenced by several physicochemical and biotic factors, equilibrium concentrations in tissues are generally related to the aqueous exposure concentration. As a result, as exposure concentrations (stressor) increase, body burden concentrations (response) also generally increase. Therefore, body burdens of the COCs in this study were interpreted to indicate the relative magnitude of exposure to those COCs even though the aqueous concentrations were below detection limits.

When the COC aqueous concentrations were below detection limits, stressor-response associations for Propositions 2 and 3 were evaluated by examining the correlation coefficients between the body burdens of COCs (in stonerollers for Proposition 2 and in stonerollers or benthic macroinvertebrates for Proposition 3) and the magnitude of the response variable of interest (e.g., percentage of fish species classified as tolerants, or benthic diversity, etc.). Rationale for this evaluation procedure is similar to the rationale presented for Proposition 1 in the preceding paragraph, which states that the body burdens of COCs provide an indicator of the relative magnitude of the exposure.

The highest aqueous mercury concentrations in EFPC were closest to the Y-12 Plant, then decreased downstream to below the detection limit at Site 4. Total mercury concentration in sediments decreased downstream from the Y-12 Plant to Site 4, but then increased at Site 5 (to levels equal to those at the site closest to the Y-12 Plant). These data indicate a clear longitudinal, downstream gradient of decreasing aqueous mercury concentrations from Site 1 to Site 4. For Proposition 1, crayfish were the only aquatic indicator organisms in this study with body burdens of mercury that were significantly correlated with aqueous mercury concentrations ($r^2=0.999$, $P<0.05$, linear regression).

However, the poor statistical correlations between aqueous mercury concentrations and mercury body burdens in both fish species examined in this study should not necessarily suggest that the stressor-responses were weak. The poor correlations were due to the body burden results from a single sampling site that deviated from the overall longitudinal trend. As previously discussed in Sect. 6.2.3.2, "Fish" subsection, the elevated body burdens of mercury for redbreast sunfish at Site 3 and for stonerollers at Site 2 were probably due to increased mercury exposure via ingestion of a contaminated food source that was specific to the particular site. The increased dose of mercury to the fish via the ingestion exposure route at these two sites could have overshadowed the mercury body burdens that were due solely to water-borne sources.

The highest aqueous zinc concentration (33.7 μg/L) in EFPC during this study was at Site 4. Aqueous zinc concentration at the site closest to the Y-12 Plant was 33.4 μg/L, and concentrations steadily decreased downstream to Site 3. Unlike aqueous mercury concentrations, zinc aqueous concentrations increased at Site 4, then steadily decreased downstream to Site 6. A clear longitudinal, downstream gradient of decreasing aqueous zinc concentrations occurred from Site 1 to Site 3. However, aqueous zinc concentrations were not statistically correlated with zinc body-burdens in any of the aquatic indicator organisms ($P > 0.05$, linear regression, $n=3$).

Of the 42 COCs that were below detection limits in surface water, 10 were present in at least one of the aquatic indicator organism body burden concentrations. These were maximum in samples collected closest to the Y-12 Plant, and then decreased downstream (Table 6.84). The ten contaminants, and the aquatic biota in which they were present in decreasing concentrations downstream from the Y-12 Plant, are as follows:

- Aroclor 1260 in stoneroller, redbreast sunfish, and crayfish;
- benzo(a)pyrene in stoneroller and crayfish;
- chrysene in stoneroller;
- DDE in stoneroller;
- dieldrin in stoneroller and crayfish;
- heptachlor in stoneroller;
- indeno(1,2,3-cd)pyrene in stoneroller;

- mercury in crayfish;
- uranium in stoneroller; and
- zinc in stoneroller and redbreast sunfish.

It is interesting that 9 of the 10 contaminants displayed decreasing body burden concentrations in stonerollers downstream from the Y-12 Plant, in comparison to 4 contaminants in crayfish, and 2 contaminants in redbreast sunfish. These data suggest a strong stressor-response association in support of Proposition 1. These data also suggest that stonerollers can be excellent in situ indicators of contaminant exposure to fish in EFPC, and offer better resolution of contaminant exposure than the redbreast sunfish.

Aqueous mercury concentrations were statistically correlated with the percentage of captured fish classified as tolerants ($r^2=0.9897$, $P<0.05$, $n=3$, linear regression), whereas aqueous zinc concentrations were not correlated ($r^2=0.9734$, $P>0.05$, $n=3$, linear regression) (Proposition 2). Aqueous concentrations of all other COCs were below detection limits. Thus, mercury was the only COC that satisfied both of the following conditions: (1) was above detection limits in the surface water and (2) displayed a strong stressor-response for supporting Proposition 2.

Five contaminant body burdens in stonerollers were statistically correlated with the percentage of captured fish classified as tolerants (Proposition 2). The five contaminants included three PAHs and two metals:

- benzo(a)pyrene ($r^2=0.9514$, $P<0.05$, $n=4$, linear regression),
- benzo(g,h,i)perylene ($r^2=0.9428$, $P<0.05$, $n=4$, linear regression),
- indeno(1,2,3-cd)pyrene ($r^2=0.9504$, $P<0.05$, $n=4$, linear regression),
- uranium ($r^2=0.9888$, $P<0.005$, $n=6$, linear regression), and
- zinc ($r^2=0.9554$, $P<0.005$, $n=6$, linear regression).

No data were obtained on the relationship between body burden of these 5 specific contaminants and subsequent toxicological effects in fish. However, the body burdens provide evidence of exposure to multiple contaminants with demonstrated toxicities to aquatic biota during aqueous exposures (a stressor). The percentage of captured fish species classified as tolerants provides

evidence that the fish community has been impacted (a response). Significant correlations between the 5 contaminant body burdens and the percentage of captured fish classified as tolerants does not guarantee a causal relationship between the variables. However, these strong correlations along with the significant correlation between aqueous mercury concentrations and percentage of tolerant fish species, provide a reasonable basis for concluding a strong stressor-response association for Proposition 2.

Evidence of stressor-response associations between COC concentrations and taxonomic richness or diversity (Proposition 3) were practically absent. For example, mercury and zinc aqueous concentrations were not significantly correlated with taxonomic richness or diversity indices for fish or benthic macroinvertebrates. In addition, no contaminant body burdens in stonerollers, redbreast sunfish, or the benthic macroinvertebrates were significantly correlated with taxonomic richness or diversity, regardless whether the regressions included all sites in EFPC or just the first three sites downstream from the Y-12 Plant. Only three contaminant body burdens were significantly correlated with taxonomic diversity. Mercury body burdens in stonerollers and redbreast sunfish, and zinc levels in stonerollers, were each statistically correlated with fish species diversity at Sites 3 to 6. The correlation coefficients were 0.9834, 0.9887, and 0.9189, respectively ($P < 0.05$, $n=4$, linear regression). Thus, the data indicate a weak stressor-response for Proposition 3.

Strength of association. With respect to Proposition 1, urban and agricultural runoff, as well as point source inputs, could potentially provide additional inputs of contaminants such as PAHs, PCBs, pesticides, residual chlorine, and some heavy metals into EFPC, thereby increasing the exposure concentration of those contaminants to aquatic biota. Although there are no point source inputs such as tributaries or effluents above Site 1 (except for the Lake Reality outfall), urban runoff increases downstream from the Y-12 Plant, as do point source inputs such as the Oak Ridge Sewage Treatment Plant effluent (see Sect. 3 for details on chemical inputs). If the urban runoff and Oak Ridge Sewage Treatment Plant effluent contained sufficient concentrations of the above-mentioned contaminants, then the body burdens of those contaminants should generally increase downstream from the Y-12 Plant, and specifically increase below the Oak Ridge Sewage Treatment Plant. The data indicate that most of the contaminant body burden concentrations in the aquatic biota decrease downstream from the Y-12 Plant. These observations suggest that the strength of association for Proposition 1 is strong, because the body burdens of most contaminants decrease downstream from the Y-12 Plant, whereas urban runoff and point source inputs increase downstream. The data also indicate that urban runoff inputs cannot

account for the body burdens of mercury or Aroclor 1260 in the fish and benthic macroinvertebrates.

Pesticide concentrations in redbreast sunfish, and PAHs in crayfish, generally increased downstream from the Y-12 Plant, suggesting inputs from sources other than the Y-12 Plant as presented in Sect. 3. Pesticide concentrations in redbreast sunfish increased downstream from Site 1 to a maximum value at either Site 3, Site 4 or Site 5, depending on the pesticide. This trend seems to indicate that an additional input of these pesticides into EFPC is occurring from runoff and probable input from the Oak Ridge Sewage Treatment Plant. PAHs in crayfish also increased steadily downstream from the Y-12 Plant, reaching a maximum concentration at Site 3. This trend seems to indicate that urban runoff or some unknown point source may be introducing additional PAHs into EFPC somewhere between Sites 1 and 3. The body burden trends for pesticides in redbreast and PAHs in crayfish should not be interpreted to indicate that the Y-12 Plant is not a source of some of these COCs that bioaccumulate in aquatic biota in EFPC because the trends in several pesticides and PAH body burdens in stonerollers clearly indicate an association with the Y-12 Plant.

Stream habitat alterations such as dredging, stream bank alterations, or impounding often have negative impacts to fish and benthic communities. These activities could physically destroy benthic communities and fish spawning areas. In addition, dredging and stream bank alterations typically cause increased siltation, which can result in reduced fish production and diversity (Berkman and Rabeni 1987). However, these activities have not been conducted extensively in EFPC except for the rerouting of a short section of the creek into a concrete channel in the early 1980s. Thus, habitat alteration does not appear to be a factor in explaining the proportion of tolerant fish species (Proposition 2) or impacts to fish and benthic macroinvertebrate taxonomic richness and diversity (Proposition 3) in EFPC below the Y-12 Plant.

Alterations in streamflow and ambient water temperatures (i.e., augmented, fairly consistent flow and elevated temperatures) could potentially have impacts to the benthic macroinvertebrate and fish communities in EFPC, especially nearer to the Y-12 Plant. Thus, the alterations in streamflow and ambient water temperatures should be considered as possible contributing factors toward impacts to fish and benthic macroinvertebrate taxonomic richness and diversity (Proposition 3).

Although ambient toxicity tests (7-d tests using *Ceriodaphnia* and fathead minnows) generally have indicated no consistent toxicological impacts from EFPC surface water, exceptions have been observed. For example, significant reductions in growth of fathead minnows have been

observed at EFK 22.8 and other sites downstream from the Y-12 Plant (Loar 1992). In situ studies from 1989 through 1991 on the growth and survival of fingernail clams in EFPC have indicated that growth of clams in reference streams was always greater than at any of the sites in EFPC (Kornegay 1992). Survival rates of clams at EFK 13.8 and the reference sites were similar all three years. However, mortality of clams at EFK 23.4 (below Lake Reality) was rapid in 1989 and 1991, but similar to survival of clams at EFK 13.8 in 1990. The results of the ambient toxicity test and in situ clam studies provide evidence of toxicological effects [consistent with impacts on the proportion of tolerant fish species (Proposition 2) and impacts to fish and benthos taxonomic richness and diversity (Proposition 3)] to aquatic biota in EFPC downstream from the Y-12 Plant. Unfortunately, the studies do not clearly identify the stressor(s) responsible for the impacts. Interestingly, mean concentrations of total residual chlorine that were measured during the 7-d media toxicity tests did not exceed the EPA Water Quality Criteria (WQC) for protection of aquatic life (11μg/L). In addition, Loar (1992) stated that although episodic increases of chlorine concentration probably affect benthos at EFK 24.4 (above former New Hope Pond), the chlorine concentrations cannot account for the low abundance and diversity at EFK 23.4. Thus, total residual chlorine is probably not a significant chemical stressor below Lake Reality.

The aqueous mercury concentrations at the three sites closest to the Y-12 Plant (Sites 1, 2, and 3) (0.54 to 0.32 μg/L) exceeded the concentrations known to elicit toxic effects in fish [$<$0.1 μg/L, 28-d EC_{50} for rainbow trout (*Salmo gairdneri*); 0.23μg/L, significant reduction in growth of fathead minnows at 30-d post hatch]. Thus, only fish species most tolerant of the high mercury concentrations would be expected to successfully live and reproduce at these sites. The high proportion of tolerant fish species at these three sites (73, 63, and 56%, respectively) provide supporting evidence that the observed mercury concentrations in surface water have resulted in toxic impacts to sensitive fish species, thereby allowing the more tolerant species to dominate the community. Concomitantly, the absence of intolerant fish species such as darters (*Etheostoma sp.*), which should be present based on the stream habitat, strengthens the evidence. In addition, the lowest EPT richness was observed at the site closest to the Y-12 Plant. This provides further evidence that water-borne contaminant releases from the Y-12 Plant have impacted the aquatic communities. Therefore, the strength of association between impacts to the fish and benthic macroinvertebrate communities in EFPC and releases of water-borne contaminants from the Y-12 Plant (Propositions 2 and 3) is strong.

Plausibility. The plausibility for Proposition 1 (water-borne COCs from the Y-12 Plant account for the largest share of the COC body burdens in aquatic indicator organisms in EFPC) is strong. Mercury and Aroclor 1260 are two contaminants that have high bioconcentration factors in aquatic organisms (Eisler 1986, 1987). Higher molecular weight PAHs, pesticides such as chlordane, dieldrin, heptachlor, and DDE, and heavy metals such as zinc, chromium, and arsenic are also known to bioaccumulate in aquatic organisms. Mercury and PCBs have been continually released from the Y-12 Plant into EFPC for many years. In addition, the data from this and historical studies (Van Winkle et al. 1984; TVA 1985; Loar 1992; Hinzman 1992) clearly indicate a longitudinal trend of bioaccumulation of other contaminants in fish and benthic macroinvertebrates downstream from the Y-12 Plant. Thus, it is apparent that the PAHs, pesticides, and other inorganic contaminants that were found in the aquatic biota were at least partially attributed to releases from the Y-12 Plant. Therefore, the plausibility for Proposition 1 is strong.

Aquatic communities that are subjected to physical and/or chemical stressors are known to undergo structural changes, with taxa that are most adaptable or tolerant to the stressor(s) becoming the predominant assemblages (Proposition 2). As a result, species diversity and the total number of taxa usually decrease (Proposition 3). Propositions 2 and 3 are plausible because the surface water mercury concentrations (0.54 to 0.32 μg/L) at the three sites closest to the Y-12 Plant exceed the concentration known to cause toxic effects in fish (<0.1 μg/L for rainbow trout; 0.23 μg/L for fathead minnows). This alone could account for the observed impacts to the fish community structure and diversity. In addition, although the surface water concentrations of Aroclor 1260 were below the detection limit (0.2 μg/L), the high body burdens observed in redbreast sunfish and stonerollers suggests that the aqueous concentrations probably exceeded the WQC for PCBs (0.001 μg/L).

Summary of aquatic weight-of-evidence analysis

This weight-of-evidence analysis evaluated three propositions, which were conclusions based on the results of the ERA field sampling, historical studies on EFPC, as well as laboratory toxicological bioassays. The propositions were evaluated against five criteria (temporal association, spatial association, stressor-response, strength of association, and plausibility). Major observations from the ERA field sampling and historical studies indicated that numerous COC body burdens in the aquatic indicator organisms (stonerollers, redbreast sunfish, crayfish, and benthic macroinvertebrates), as well as several measures of community structure (fish and benthic macroinvertebrate taxonomic richness and diversity, and percentage of fish species classified as tolerants) showed the greatest impacts closest to the Y-12 Plant, then decreased downstream in

EFPC. In addition, COC body burdens and community impacts in biota from all sites in EFPC usually were greater than equivalent impacts at a reference site.

The data for mercury and PCBs indicate a clear temporal association between the release of these two contaminants from the Y-12 Plant and their subsequent bioaccumulation in aquatic biota downstream in EFPC. Several variables (such as the percentage of captured fish species that are classified as tolerant of degraded water quality conditions, fish and benthic macroinvertebrate taxa richness, and species diversity) have consistently indicated that aquatic biota in EFPC are adversely impacted in comparison to biota in a reference stream. Also, BMAP data show a recovering ecological system in the upper part of EFPC, below Lake Reality.

Spatial associations were strong for all three propositions based on current and historical evidence. Body burdens of many COCs were greatest in biota collected closest to the Y-12 Plant, and decreased downstream. Also, the COC body burdens in biota from most sites in EFPC were greater than the body burdens in biota from the reference site. Similar trends were observed for the percentage of tolerant fish species (Proposition 2). Fish and benthic macroinvertebrate taxonomic richness and diversity (Proposition 3) generally increased downstream from the Y-12 Plant, indicating that the most severe impacts occurred closest to the plant.

Stressor-response associations were evaluated by determining linear correlation coefficients between estimated COC exposure concentrations (the stressor) and the magnitude of some response variable [the response (such as percentage of fish classified as tolerants)]. In the absence of COC concentration data in surface water, COC body burden concentrations were used as an indicator of the stressor exposure level.

Mercury in surface water was significantly correlated with mercury body burdens in crayfish. Of the COCs that were below detection limits in surface water, ten were present in at least one of the aquatic indicator organism body burden concentrations that were maximum in samples collected closest to the Y-12 Plant, then decreased downstream. Mercury concentrations in surface water were correlated with the percentage of fish classified as tolerants, but zinc concentrations in surface water were not (Proposition 2). Five contaminant body burdens in stonerollers were correlated with the percentage of fish classified as tolerants. Evidence of stressor-response associations between COC concentrations and taxonomic richness or diversity (Proposition 3) were practically absent. Thus, the data suggest a strong stressor-response association for Proposition 1, a moderate association for Proposition 2, a weak association for Proposition 3.

Strength of association for all three propositions was strong. Likewise, plausibility for all three propositions was strong. The COCs from this study are known to bioaccumulate in most aquatic organisms. Although most COC concentrations in surface water were below detection limits, the large number of COC body burdens displaying longitudinal decreases in concentration downstream from the Y-12 Plant suggests that the Y-12 Plant is the source of the COCs in the biota (Proposition 1). Propositions 2 and 3 are plausible because the surface water mercury concentrations (0.54 to 0.32 μg/L) at the three sites closest to the Y-12 Plant exceed the concentration known to cause toxic effects in fish (<0.1 μg/L). This alone could account for the observed impacts to the fish and benthic macroinvertebrate community structure and diversity.

The conclusions from the weight-of-evidence analysis have implications for the assessment endpoints associated with Policy Goals 4 (a fish community indicative of undegraded conditions) and 5 (no adverse effects from contaminants to aquatic organisms and/or predators that feed on them). For example, aqueous mercury concentrations in EFPC have been observed that exceeded levels known to produce toxicological impacts to sensitive (e.g., $<0.1\mu$g/L in rainbow trout) as well as tolerant species (e.g., 0.23 μg/L in fathead minnows). This alone could affect community structure (Policy Goal 4 and its assessment endpoint), due to increased proportion of tolerant fish species (Proposition 2) and impacts to taxonomic richness and diversity (Proposition 3). Elevated aqueous mercury concentrations that exceed AWQC also place fish and benthic macroinvertebrate communities at risk, which links to Policy Goal 5 and assessment endpoint 5a. Elevated body burdens of mercury and other contaminants continue to place aquatic organisms and their predators at risk (Policy Goal 5, assessment endpoints 5b and 5c). The aquatic weight-of-evidence analysis indicates that Policy Goals 4 and 5 are not being met, most likely due to ongoing releases of contaminants from the Y-12 Plant.

6.4.1.3 Terrestrial findings

A weight-of-evidence treatment is applied to terrestrial findings below.

Propositions

The weight-of-evidence analysis for the terrestrial biota makes the following propositions concerning EFPC soil contaminants. Common to both propositions is the premise that EFPC soil contaminants were released from the Y-12 Plant and were transported by and deposited from EFPC surface water. The propositions are:

(1) EFPC soil contaminants have resulted in elevated body burdens of these contaminants in terrestrial organisms residing on the floodplain, as indicated by field-observed measurements of earthworms, small mammals, and birds.

(2) EFPC soil contaminants have resulted in reduced abundance of terrestrial organisms residing on the floodplain, as indicated by field-observed measurements terrestrial insects, especially 10 families of insects that live in or on surface soils or herbaceous vegetation.

The propositions explicitly address a subset of the EFPC terrestrial community – the animal indicator species. These species are generally ubiquitous and, for one or more of their life stages, relatively sessile inhabitants of the floodplain. The 10 families of insects considered in detail in the following analysis are inhabitants of soil, soil surface, or herbaceous vegetation, or are predators of other such inhabitants (Borros and DeLong 1964). The propositions link directly to Policy Goal 7 (Sect. 6.1.3) – the protection of terrestrial animals and their predators from the effects of contaminants. The propositions link indirectly to Policy Goal 2 because terrestrial organisms represent an essential resource for migratory birds passing through or residing seasonally on the floodplain.

Mercury is the principal contaminant considered in the weight-of-evidence analysis for several reasons:

- Mercury is the primary chemical contaminant of concern in EFPC.
- The only known source of mercury is the Y-12 Plant.
- There are more toxicity data on mercury than on other contaminants.
- Surface soil mercury concentrations along the creek bank generally decrease with distance from the Y-12 Plant.

Additional evidence for these propositions is provided by data on other contaminants such as the metals, cadmium, and uranium; the PCB, Aroclor 1260; PAHs; and the pesticide, Chlordane.

Support for these propositions is presented in the following subsections.

Analysis

This weight-of-evidence analysis for the terrestrial community considers 4 of the 5 criteria used in the analysis for the aquatic community: spatial association, stressor-response, strength of association, and plausibility. There is no known historical information on terrestrial communities

at EFPC upon which to base an argument for a temporal association prior to Y-12 start-up. Therefore, temporal association is not considered.

Spatial association. Proposition 1 is supported by a clear spatial association between body burdens of mercury in earthworms (*L. terrestris*) and creek-bank soil-mercury concentrations. Maximum mercury concentrations in the surface soil samples nearest the banks of EFPC decreased from 1170 mg/kg near the Y-12 Plant to 216 mg/kg at the confluence with Poplar Creek. Body burdens of mercury in earthworms and soil mercury concentrations at biotic sampling sites decreased with increasing distance from the Y-12 Plant; the only exception is Site 1, which has a narrow floodplain. Body burdens of mercury in earthworms decreased from a maximum of 33 mg/kg at Site 2 to 4.7 mg/kg at Site 6.

The two highest body burdens measured in mice (*P. leucopus*) were for individuals captured at Site 2, which is at the highly contaminated NO site. Otherwise, there was not a strong association between soil mercury concentrations at biotic sampling sites and mice body burdens. Biotic sampling sites would not necessarily be expected to show a trend of decreasing soil contaminant concentration with increasing distance from the Y-12 Plant. These sampling sites were chosen because they had high mercury concentrations, as determined in historical and Phase Ia studies (Radian 1993a). These "hot spots" are thought to have accumulated sediments transported downstream and deposited by past floods – sediments released over decades or more and coming from stream segments of varying length.

Measured body burdens in earthworms almost certainly reflect local surface soil contamination levels more accurately than do body burdens in mice. Earthworms feed on decomposing surface litter, may drag leaves into their burrows in the upper soil layers, and ingest soil while burrowing (Barnes 1974). Earthworm samples analyzed for contaminants included whatever soil was in the earthworms' digestive tracts at the time of collection (in order to provide measurements of exposure to worm predators). To the extent that measured body burdens were due to the soil contents, they reflect mercury levels in floodplain surface soils near to where the worms were sampled. To the extent that measured values were due to actual tissue body burdens, they more likely reflect integrated exposures to mercury over an area of many square meters. An area this size is still likely to be considerably smaller than the home range of mice, which is ~0.1 to 0.6 ha (0.25 to 1.5 ac) (Ambrose 1975; Burt and Grossenheider 1976).

Body burdens of mice are more likely a result of exposures to contaminants over a much larger area of floodplain where the samples were taken. This may explain why mice from Site 2 had higher overall contamination than mice from Site 1, upstream. At Site 1, the floodplain is

narrow. At Site 2, where the floodplain is larger, where sediment deposition by floods has been greater, or both, more mercury may be available mice from other "hot spots" downstream of the NO site (Site 2) [e.g., the BR site (near Site 3)] may not show exceptionally high body burdens of mercury, because the mercury is located in the soil at depths that reduce the likelihood of exposure (Sect. 3.2.5.2). At both the NO and BR sites, maximum concentrations are found below 16 cm (6 in). Surface concentrations at the NO site are high, 451 - 647 ppm in the top 11 cm (4 in) (Fig. 3.88). At the BR site, surface concentrations of mercury are low in many areas, with shallow "spikes" at 11 - 16 cm (4 - 6 in) (Figs. 3.86, 3.87). Maximum mercury concentrations in the soil at the Grand Cove soil-sampling site (53 ppm), which is between biotic sampling Sites 4 and 5, occur even deeper, with no appreciable contamination above 38 cm (13 in) (Fig. 3.85).

Additional evidence in support of Proposition 1 is the elevated body burdens of contaminants in small mammals, earthworms, and insects as compared to those from the reference site. Cadmium and zinc in all mice captured at all EFPC sites; antimony in six mice; and arsenic, cadmium, selenium, uranium, zinc, and, of course, mercury, in earthworms at all sites exceeded the reference site measurements (Tables 6.27, 6.35). Chromium levels in earthworms at Sites 1 and 6 were similar to those at the Mill Branch reference site, but at all other EFPC sites were greater. Body burdens of Aroclor 1260 in mouse homogenates at all sites in EFPC exceeded those from the reference site (Table 6.28). Body burdens of 6 metals, Aroclor 1260, and some pesticides in insects from between 2 to 5 EFPC sites exceeded those from the reference site (Tables 6.31 through 6.33); cadmium and uranium were below detection levels at all sites.

The association between elevated body burdens in earthworms and their predators links the terrestrial weight-of-evidence analysis for Proposition 1 to Policy Goal 6: the maintenance of terrestrial animals with abundances and distributions indicative of undegraded conditions. Body burdens of mercury and other contaminants in EFPC earthworms (arsenic, cadmium, mercury, selenium, uranium, and zinc), which were elevated relative to body burdens in earthworms at the reference site, are elevated in the few individual shrews and wrens captured on EFPC, but not in the mice. This is expected because shrews and wrens are more likely to consume earthworms and insects than are mice. Body burdens of arsenic (344 μg/kg), cadmium (5949 μg/kg), mercury (1911 mg/kg), and selenium (934 mg/kg) in the shrew captured at Site 4 were greater than that of the composite body burdens of mice from the same site ($<$ 179, $<$ 1536, $<$ 229, and 314 μg/kg, respectively). The wren captured at Site 4 had body burdens of cadmium (4669 μg/kg), mercury (3550 μg/kg), selenium (1039 μg/kg), and zinc (42168 μg/kg) that were greater than those of the mice at that site; the body burden of zinc in mice at the site was 32713 μg/kg. Similarly, the wren from Site 2 had a body burden of mercury (3486 μg/kg) that

exceeded that of the two mice at Site 2 with body burdens above detection limits (i.e., 675 and 1105 μg/kg). Further linkage to Policy Goal 6 is provided by the knowledge that terrestrial predators such as owls, hawks, and foxes feed predominantly on small mammals and birds.

Many year-round resident or migrant birds that nest and raise their young on the EFPC floodplain during the summer (approximately 34 species) depend primarily on small invertebrates for food during this critical period and, thus, may be exposed to contaminants by ingesting earthworms and insects. Wrens are insectivores, and the wrens captured on the EFPC floodplain showed high body burdens of metals that were elevated in earthworms or insects with terrestrial juveniles (i.e., antimony, arsenic, cadmium, chromium, mercury, selenium, and zinc). Bioaccumulation of contaminants in birds would cause body burdens to increase through time. These results link Proposition 1 to Policy Goal 2, the protection of migratory birds. Reductions in insect abundance would also be detrimental to nesting insectivorous birds, thereby potentially linking Proposition 2 to Policy Goals 2 and 6.

The spatial association for Proposition 2 is supported by the inverse relationship between soil-mercury concentrations and insect abundance. The total number of insects with terrestrial juveniles that were captured, the number captured per trap-night, and the abundances of 10 families (2 Coleoptera, 5 Lepidoptera, and 3 Homoptera), representing a majority of those captured, generally increased with increasing distance from the Y-12 Plant (Table 6.64). Creek-bank soil-mercury concentrations, on the other hand, decreased with increasing distance from the Y-12 Plant. For example, the total number of insects captured ranged from 1119 at Site 2 to 4001 at Site 6 in the following rank order: 6, 5, 4, 3, 1, 2. The rank order of mercury body burdens in earthworms (and PCBs in insects) from these sites is exactly the opposite: 2, 1, 3, 4, 5, 6 (Fig. 6.54). The general trend of increasing abundance with increasing distance from the Y-12 Plant was also seen in the Unidentified Coleoptera and Unidentified Lepidoptera. The abundances at the Hind's Creek reference site of 9 of the 10 insect families were greater than abundances at one or more of the most upstream EFPC sites, but there were no other consistent relationships between EFPC insect abundance data and those data from the reference site.

The 10 insect families, which dominated insect samples from both EFPC and the reference site, are the kinds of insects that would be expected to have high exposures to contaminants deposited on floodplain soil surfaces or low lying vegetation. These families were from 3 orders of arthropods. Carabid beetles (Carabidae) and rove beetles (Staphylinidae) are coleopterans; leafhoppers (Cicadellidae), planthoppers (Flatidae), and jumping plant lice (Psyllidae) are homopterans; and inchworm moths (Geometridae), noctuid moths (Noctuidae), prominent moths (Notodontidae), snout moths (Pyralidae), and tortricid moths (Tortricidae) are lepidopterans.

Adults of both of the coleopteran families are generally predators of other insects and are found in the soil and under bark or decaying plant matter and debris. Coleopteran (beetle) larvae (i.e., grubs) are generally vermiform and subterranean. Homopterans are specialized herbivores that spend their entire life on the surface of herbaceous vegetation. Many lepidopteran larvae (caterpillars) eat leaves and crawl about on the surface of soil, litter, and vegetation. Therefore, insects from these 10 families would, in general, be more likely to be exposed to contaminants deposited by floodwaters on floodplain soil surfaces or low lying vegetation than would insects that hatch from eggs laid on leaves or bark in the tree canopy and spend their adult lives there.

Stressor-response. There is evidence for a relationship between the concentration of mercury in EFPC soils and body burdens in earthworms, small mammals, and birds (Proposition 1) provided by published toxicological literature. Revis et al. (1989b), using EFPC soils and sediments, found that mercury concentrations in mouse livers and kidneys were highly correlated with diet mercury concentrations. Mice probably do not consume significant amounts of contaminated soil or invertebrates and, therefore, are not in a pathway to show effects. Carnivorous shrews and wrens do consume soil-dwelling invertebrates and, therefore, are in the pathway to show effects. Thus, these results indirectly support the proposed causal relationship between soil mercury concentrations via food and the observed body burdens of mercury in shrews and wrens.

Published dose-response data for annelids can be taken as weak indirect support for a causal relationship between the increasing abundance of at least 2 of the 3 most abundant arthropod orders (Coleoptera and Lepidoptera) on the EFPC floodplain and the observed pattern of soil-mercury concentrations, which generally decrease with distance from the Y-12 Plant (Proposition 2). Eisler (1987) reviewed evidence for the causal relationship between soil mercury concentrations and mortality of soil dwelling worms. A soil concentration of 25 mg methylmercury/kg was fatal to all tiger worms (*Eisenia foetida*) exposed for 12 weeks; at 5.0 mg/kg, 21% died at 12 weeks. Inorganic mercury at 0.79 mg/kg soil produced 50% mortality of the earthworm (*Octochaetus pattoni*); 5.0 mg/kg produced 100% mortality. Annelid worms and the larvae of some coleopterans and lepidopterans share a vermiform body shape that increases the likelihood of coming into direct contact with contaminated vegetation or soils. Mercury levels in earthworms (and their gut contents) from these sites range from 5 to 33 mg/kg, suggesting that lethal exposures are possible at these sites, where average soil concentrations range from ~10 to 400 mg/kg (Figure 6.76).

Additional evidence for a stressor-response relationship is provided by composite insect body burdens and insect abundance. The Cicadellidae were the most abundant homopterans and, by

far, the most abundant terrestrial-origin insect family at all sites, comprising between about 13 and 42% of all individuals sampled (Table 6.64). This family had its lowest abundances at Sites 2 and 3, where composite insect body burdens of mercury were highest (196 and 3189 μg/kg, respectively). There were 196 (about 18%) and 256 (13%) individuals of Cicadellidae at Sites 2 and 3, compared to a range of 488 (32%) at EFPC Site 1 to 1547 (41%) at the Hinds Creek reference site. These data do not suggest that contaminant body burdens of the magnitude measured in *composite* samples will cause mortality of any particular group of insects, because insects of varying exposures and body burdens were combined. Nevertheless, some insect groups inevitably had above average body burdens of mercury and other contaminants at these two sites. It has already been postulated that the 10 most abundant families on EFPC would be expected, in general, to have higher exposures than other insects. If the Cicadellidae were one of the groups with above average body burdens, it may explain their relatively low abundances at these two sites.

Strength of Association. There is a moderate to strong association between EFPC soil contaminants and body burdens of mercury in animals living in or on the soil surface (Proposition 1) and abundances of insects with terrestrial juveniles (Proposition 2). In general, EFPC floodplain contaminants are the only reasonable source of many contaminants to terrestrial biota. As previously noted, there is no other identified source of mercury in the EFPC watershed. Mercury concentrations in surface soils at sampling stations on transects along the entire length of EFPC generally decrease with distance from the banks of EFPC (Figures 3.78-3.80), as would be expected if the primary source of mercury in surface soils was EFPC surface water overflowing the bank. For other contaminants, the association is not as strong because of the possibility of additional sources along the EFPC floodplain (e.g., the Oak Ridge Sewage Treatment Plant).

The strength of the association hypothesized in Proposition 2 is strongly supported by distribution patterns of a majority of the insects with terrestrial juveniles that were sampled on EFPC. In 5 of 10 families plus the unidentified Lepidoptera, there was a significant difference ($P < 0.05$) between ranks of upstream sites (e.g., Sites 1, 2, and 3) and downstream sites (e.g., Sites 4, 5, and 6). Combined, the 10 families of coleopterans, homopterans, and lepidopterans that increase in abundance with distance from the Y-12 Plant represent between about 48 and 70% of all insects with terrestrial juveniles captured at a site. The Unidentified Coleoptera and Unidentified Lepidoptera, which also show this trend, represent an additional 13 to 24% of total site captures. Thus, the total number of individuals in groups showing a spatial association with mercury contamination represent between 70 and 84% of all terrestrial insects captured.

Changes in the abundances of terrestrial insects along EFPC could be a result of species, having different temperature, pH and desiccation tolerances, life habits, and resource requirements, replacing one another in response to changes in ecological conditions that are not associated directly with chemical contamination, such as vegetational changes. Soil and climatic variables, however, are unlikely to vary greatly in a systematic fashion in the 22 km (13 mi) stretch of EFPC, and there is no evidence that vegetation changes significantly over this distance (Table 6.67). Furthermore, the fact that just three orders uniformly dominate the terrestrial insect fauna along the entire length of EFPC – these three orders also dominate the reference site – strongly suggests that natural species replacements occur within and not between orders. Species in other orders do not decrease in abundance as these three orders increase; rather, they show no trend. The increase in abundance with increasing distance from the Y-12 Plant exhibited by the dominant families from these three orders is strong evidence that one or more stressors associated with EFPC diminishes with increasing distance downstream.

Plausibility. The plausibility of Proposition 1 is high. There are no known sources of mercury to EFPC other than the Y-12 Plant. If there were unidentified sources, floodplain surface-soil mercury concentrations nearest the creek would probably not decrease with increasing distance from the Y-12 Plant but would show high concentrations downstream of Site 2. For example, PCBs show a peak at Site 3, which is immediately downstream of an electrical transformer substation. According to Eisler (1987), mercury bioconcentrates in invertebrates (BCF = 100,000) and adsorbs highly to soil organic matter. Thus, mercury is accessible to earthworms and larval insects living in and on EFPC soil. It is also likely that the metabolisms of earthworms and insects are different from that of mice, which did not efficiently absorb ingested mercury (Revis et al. 1989b).

The plausibility of Proposition 2 is moderate to high. The majority of insects with terrestrial juveniles that were captured had lower population sizes where surface soil mercury concentrations were highest. The 10 families of insects showing this pattern are inhabitants of soil, soil surface, or herbaceous vegetation, or are predators of other such inhabitants (Borros and DeLong 1964) and would be expected to have high exposures to contaminants deposited on the floodplain soil surface or low lying vegetation. It is plausible that contaminants from the Y-12 Plant, such as mercury, which can bioconcentrate in invertebrates to levels that could cause behavioral alteration or reproductive impairment, are responsible for the observed abundance patterns.

Summary of terrestrial weight-of-evidence analysis

The terrestrial weight-of-evidence analysis evaluated two propositions about the relationship between Y-12 Plant-associated contaminants on the EFPC floodplain and ecological effects on the terrestrial floodplain biota. Evaluated against four criteria (spatial association, stressor-response, strength of association, and plausibility), the weight-of evidence supports these two propositions. It is concluded that increased body burdens of mercury and other contaminants in earthworms, mice, shrews, and wrens result from EFPC soil contaminants that originated at the Y-12 Plant and were transported by and deposited from EFPC surface water onto the floodplain. It is also concluded that the decreased abundances of 10 families of insects with terrestrial juveniles at EFPC biotic sample sites nearest the Y-12 Plant (especially Sites 1, 2, and 3) result from EFPC soil contaminants.

These conclusions have implications for the assessment endpoints. Elevated body burdens of EFPC contaminants in earthworms and their predators put still other unsampled and unanalyzed terrestrial predators on the EFPC floodplain at risk from exposure to these contaminants. Reductions in insect abundances may decrease the carrying capacity of EFPC for numerous species of insectivorous birds that raise their young on the floodplain in the summer. Combined with elevated body burdens of contaminants in insects, this represents an even more potentially serious risk to bird populations. Thus, Policy Goals 2 and 6, the protection of migratory birds and the maintenance of terrestrial animal communities indicative of undegraded conditions, are probably not being met as a result of EFPC soil contaminants that originated at the Y-12 Plant and were transported by and deposited from EFPC surface water onto the floodplain.

6.4.1.4 Spatial distribution of ecological risk

The floodplain was organized into nine risk assessment segments (Fig. 6.78). These segments were developed based on the similarities of the distribution of contaminants and land-use. A more descriptive explanation of the criteria for segment selection is found in Sect. 5. Segments 1, 2, and 3 are located in the upper part of the creek, below Lake Reality, whereas Segments 4,5,6,7, and 8 are located further downstream along the east-west axis of the stream. The longest segment—9—covers approximately the last 25 percent of the EFPC floodplain, and its western edge is located at the confluence with Poplar Creek. The same risk assessment segments are being used in both human health risk and ecological risk in this RI.

Ecological risk differs from segment to segment along the ~22 km (14 mi) course of EFPC. Risk varies due to trends in exposure to contaminants and as measured by comparing contaminant body burdens relative to protective criteria (quotients) and weight-of-evidence discussions on

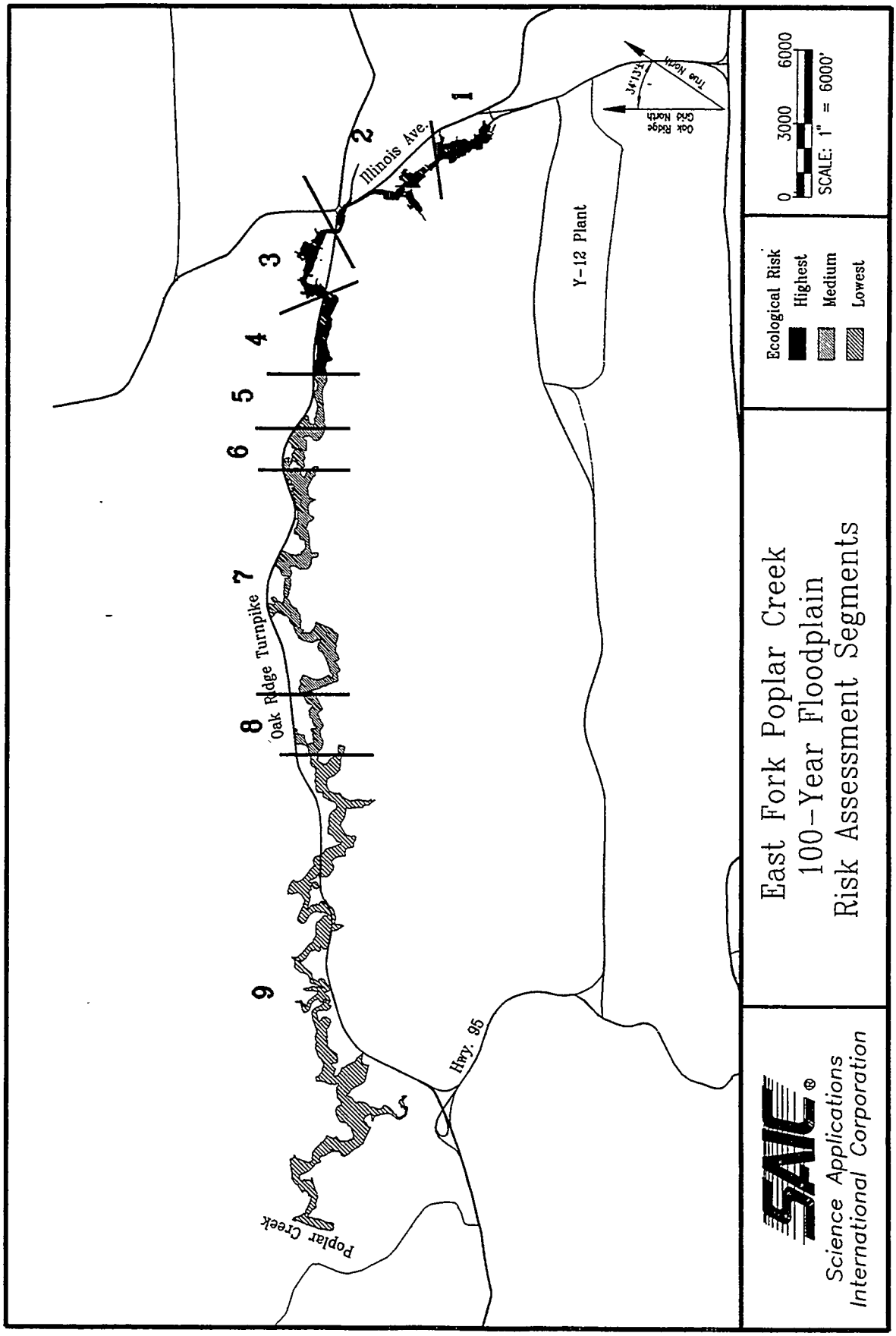

Fig. 6.78. East Fork Poplar Creek 100-year floodplain risk assessment segments.

spatial associations. Based on the trend that most contaminant body burdens, ecological community effects, and risk quotients decrease downstream from the Y-12 Plant, ecological risk is highest in Segments 1, 2, 3, and 4; these segments are closest to the source of the contamination. Also, there are several contaminated wetlands with resident invertebrates, amphibians, and other animals in these segments. Ecological risk is lowest in Segment 9, which is the farthest segment downstream from Y-12; contaminant body burdens and other assessment endpoint measures are also lowest in Segment 9. Ecological risk is intermediate in Segments 5, 6, 7, and 8, which are located in the midstream portion of EFPC

Along this overall gradient of ecological risk and within each segment, two types of ecological resources are at risk: aquatic and terrestrial. Aquatic resources—fish, benthic macroinvertebrates, and primary producers—are at highest risk because they live in the water and are exposed continuously via body contact. The highest exposures occur in the highly contaminated aquatic area below Lake Reality. Risks to aquatic biota are greatest immediately downstream from the Y-12 Plant (EFK 20-23). Some locations in the creek provide contaminated prey to fish-eating predators. Exposures to aquatic and piscivorous biota decrease downstream. Aquatic ecological resources exhibit a greater risk segment by segment than do the terrestrial resources in that particular segment.

As indicated above, terrestrial resources—birds, small mammals, worms, arthropods, and vegetation—exhibit less risk and less definitive trends than the aquatic resources for a particular segment. Exposures to terrestrial animals are primarily via contaminated food, in which contaminant levels are related to soil contaminant concentrations. Initial work shows that terrestrial areas of highest mercury concentrations are sometimes found in the bottomland hardwood forests, where flooding is most likely to have occurred. The systematic and rigorous associations among soil contamination patterns, habitats, and field-observed ecological effects will be developed for the FS/EIS activities. Yet, as explained in the risk quotient and terrestrial weight-of-evidence sections, available terrestrial measurements show trends. These trends consist of decreasing contaminant body burdens and more "normal" arthropod community structures as distance increases from the Y-12 Plant. Thus, both the aquatic and terrestrial organisms in the EFPC environment are at differing risks as a function of distance from the Y-12 Plant. In turn, these ecological risks are in three large patterns: highest in Segments 1, 2, 3, and 4 (upper reach); intermediate in Segments 5, 6, 7, and 8 (middle reach); and lowest in Segment 9 (lower reach).

6.4.2 Future Exposures and Risks

Many future exposure scenarios are possible. If there is no remediation at either the Y-12 Plant or the EFPC floodplain, exposures of the biota will not change significantly. Cessation of releases from routine Y-12 Plant operations would likely reduce chronic exposures in EFPC, but releases into EFPC by stormwater runoff from the Y-12 Plant into EFPC would likely continue. Revisions in Y-12 Plant operations that caused a reduction in the volume of water but not contaminants flowing into EFPC could increase exposure concentrations to aquatic biota. Closure of the Y-12 Plant would alter the baseflow of EFPC, causing disruption of many biotic communities, especially in the upper reaches of the creek. Because ongoing releases of contaminants from the Y-12 Plant into EFPC dominate the effects on aquatic biota in EFPC, it is not clear what baseline contaminant body burdens would be if the EFPC floodplain and sediments were the only source of contaminants to surface water.

Sediment will also continue to be a source of contaminants to the water column if the contaminant concentration in the water is below the sediment equilibrium concentration. Potential contributions of sediment mercury to surface water can be estimated by using data from the mesocosm study (Appendix Q). The study was designed to mimic flow rates in Lake Reality. Therefore, the rate at which clean surface water (groundwater in that study) accumulates mercury from the contaminated sediment can be used as a proxy flux rate for instream sediments. Water flowing at $\sim$4.0 L/min over a sediment surface of 4.68 m^2 at $\sim$100 ppm mercury showed an average increase of 17 ng mercury/L. This is equivalent to a flux rate of $\sim$1 ng/m^2/min. At the nominal baseflow rate of 5×10^6 gal/day (1.39×10^4 L/min) and assuming that the total creek surface is underlain by contaminated sediment, the calculated concentration of mercury in surface water at the end of Segment 1 (0.9 ha or 9000 m^2) would be $\sim$0.6 ng/L (0.0006 μg/L), $\sim$0.05 times the AWQC. Concentrations would increase with distance downstream as more contaminated sediment contributes to the surface water, but never above the sediment equilibrium concentration. Therefore, it is unlikely that mercury derived from sediment would ever constitute a risk to aquatic biota in the upper portions of EFPC.

To estimate surface water concentrations of sediment-derived mercury in the lower portions of the creek, additional inputs of water as well as of mercury must be considered. Baseflow increases with increasing distance downstream to more than 3.3×10^4 L/min at the confluence of EFPC with Poplar Creek (Sect. 2.6). The total area of aquatic habitat, and thus the assumed area of creek sediment, is 23.4 ha (2.34×10^5 m^2), yielding a calculated concentration of less than 0.007 μg/L at the downstream end of the creek. Because a portion of the stream bed, especially in riffle areas, has little sediment, this concentration is a very conservative estimate. It also

applies only to high sediment concentrations, since flux rate is related to concentration in the sediment. Therefore, it appears that sediments currently present in EFPC are not a potential source of exposures above the AWQC to aquatic biota in the surface water column.

Site 4 probably represents the maximum exposure to releases from the municipal sewage treatment facility, transport of spills from the commercially developed reaches of EFPC, and runoff from the remainder of the upper portion of the watershed. In addition, a portion of the stormwater runoff from outside the EFPC watershed is discharged into the municipal sewage treatment plant. At Site 4, organic analytes, mercury, and all other metals except zinc were not detectable in surface water. Concentrations of some inorganic contaminants, including some radionuclides, increased in sediments just downstream from the treatment plant outfall (Sect. 3). Mercury and PCB concentrations in aquatic biota at this site were below criteria for toxicological effects. However, mercury levels in aquatic biota exceeded the criterion for protection of piscivorous animals, and the mercury body burdens in wren, shrew, and earthworms were above the criterion for terrestrial animals. Therefore, it appears that, in the absence of releases to EFPC from the Y-12 Plant, some risk to piscivorous animals and terrestrial predators will remain as a result of other contaminant sources.

In the future, terrestrial organisms would continue to be exposed to contaminated soil in EFPC. The elevated contaminant body burdens of earthworms, small mammals, and insectivorous birds (e.g., wrens) would continue to place these populations and at risk of adverse effects. The abundance of insects with terrestrial juveniles at sites close to the Y-12 Plant would continue to be suppressed relative to downstream sites and reference sites. The potential consequences on other ecosystem components (e.g., vegetation, predators) of these changes in abundance of insects or elevated body burdens in prey organisms are discussed below.

Terrestrial predators would continue to be exposed directly and indirectly to contaminants in EFPC sediment. The dominant mode of exposure to sediment contaminants for terrestrial predators is consumption of aquatic or terrestrial organisms that are themselves contaminated. The trophic pathways from sediment to resident and migrant terrestrial predators that prey frequently on aquatic biota (e.g., raccoons, mink, and several types of birds, including raptors, great blue herons, and kingfishers) are numerous and represent the most significant exposure pathway for those predators.

In the large area of undeveloped bottomland hardwood forest, there will be a continued exposure of terrestrial animals to concentrations of contaminants sufficient to produce elevated body burdens in earthworms and their predators and reduced abundances of certain insects.

These exposures will continue via all existing pathways. The evidence reported in the ERA suggests that the dominant pathways of exposure to those terrestrial organisms showing effects of EFPC contaminants are direct contact with and ingestion of soil (for earthworms and insects) and ingestion of contaminated biota (for mice, shrews, and wrens). No data were collected for terrestrial predators, but it is likely, per food web analysis, that they will continue to be exposed to contaminants through ingestion of prey with elevated body burdens. The significance of these exposures for terrestrial predators is reflected in the ratio of average body burdens of mercury in prey organisms to the criterion for protection of terrestrial predators (0.05 mg/kg). The risk quotients for terrestrial predators consuming earthworms and insects at all EFPC sample sites range from 2 for insects at Site 6 to 664 for earthworms at Site 2 (Table 3.33). "Top" predators of small mammals and birds would be at less risk because these "top" predators forage outside the contaminated zones of the EFPC floodplain, where, presumably, exposure is lower.

Other exposure pathways from soil to terrestrial biota most likely do not individually present significant additional risk of adverse effects. The combined risk of these pathways may, however, be a significant fraction of the total risk. By itself, ingestion of water on the EFPC floodplain is probably a minor pathway of exposure for terrestrial animals. Terrestrial insects showing reduced abundances in the upstream segments of EFPC may also be exposed by ingestion of contaminated plant products, or contaminated particles deposited on plant surfaces, but neither pathway is likely significant by itself. There is some evidence to suggest plants (e.g., browse, garden vegetables, and herbaceous vegetation) do not take up significant amounts of mercury from soil (Van Winkle et al. 1984, Gist 1987, Tables 6.37 and 6.38). Compared to undeveloped areas, exposures to contaminants in soils in developed areas are probably not significant because populations of terrestrial organisms do not reside or forage to a significant degree in some areas. This exposure may increase if the currently buried lenses of mercury were to be eroded by meanderings of the creek.

Contaminated soils would continue to contribute to elevated body burdens of contaminants in terrestrial animals whose nesting or foraging habitats include pasture land, old fields and crop fields on the EFPC floodplain, and predators on these animals. Ingestion of contaminated biota and, to a lesser extent, ingestion and direct contact with soils are likely the most important pathways of exposure to the white-footed mouse and other granivorous and insectivorous small mammals and birds. The mosaic of woodlands and open fields also enhances the probability that resident and migrant birds and predatory birds, such as owls and hawks, are exposed to contaminated prey captured on open lands. Earthworms in actively cropped fields may represent an important resource to small birds and mammals. Earthworm-contaminant body burdens in currently farmed areas, where there have been historical deposits of contaminated sediment during

floods, can be elevated over reference sites if tilling practices mix soil contaminants evenly across soil depths. This situation could contribute significant exposure of terrestrial predators to EFPC soil contaminants. On the other hand, there is little reason to expect that contaminants in agricultural soils contribute significantly to the reduction in EFPC floodplain-insect in population sizes.

In residential soils and the SLB soil, contaminants will continue to make relatively minor contributions to contaminant body burdens in terrestrial animals (e.g., earthworms and their predators) because fewer organisms will be residing and foraging in these areas. The contribution of this soil to contaminant-induced reductions in insect abundance is also likely to remain insignificant. Thus, there is little reason to expect soil contaminants in residential areas to contribute significantly to the overall risk of adverse ecological effects to terrestrial biota. Organisms frequenting these developed areas (e.g., starlings) are most likely foraging over a wide area of similar habitat that is not contaminated. Without additional evidence indicating that urbanized species preferentially forage or reside at contaminated sites, it is reasonable to conclude that their expected exposure to contaminants is insignificant.

In summary, the future would result in the continued exposure of: (1) aquatic biota to direct contact and ingestion of EFPC surface water, food, and sediment, (2) aquatic predators preying on other contaminated aquatic organisms or terrestrial insects, (3) terrestrial animals dwelling in or consuming contaminated soil, and (4) terrestrial predators preying on contaminated aquatic or terrestrial animals in the EFPC floodplain. The continued exposures of aquatic and terrestrial biota and their habitats to contaminants would result in continued elevated body burdens of contaminants in these biota and risk to their predators, along with impacts to community structure and taxa diversity.

Wetlands in contaminated areas of the floodplain would continue to receive contaminants transported by storm flow. Contaminated soils could act as a source of contamination to wetlands in the watershed. Contaminated soils could be eroded and transported as suspended load in the high flow of flood events. As flow velocity decreases and flood waters recede, contaminants transported as suspended solids drop out of suspension and are redeposited in wetlands in another part of the floodplain. Because wetlands in the EFPC floodplain have formed in low-lying areas of the floodplain subject to deposition, the wetlands could receive future contaminants. In this manner contaminants could migrate and relatively uncontaminated soils in the floodplain downstream from the source area could become contaminated. In the future, contaminated soils in wetlands would remain a potential source of contamination to numerous other receptors, including soil, sediment, surface water, groundwater, plants, animals, and humans.

The floodplain contains trees, some of which have cavities and other hiding/brood places for bats. It is likely that these trees will remain and provide potential habitat to threatened and endangered bat species. However, the endangered Indiana bat has major population centers in Missouri and Kentucky and it is not likely to reach Eastern Tennessee. The endangered gray bat is also concentrated in cave regions; they are mostly found in Arkansas, Missouri, Kentucky, Tennessee, and Alabama and forage primarily along rivers or lake shores. It is not likely that this bat will expand its range to the small stream area of EFPC.

6.4.3 Uncertainties

This section presents a discussion of sources of uncertainty in the baseline ERA for EFPC and the relative influences of the uncertainties on the evaluation of risks to ecological resources (animals, plants, and habitats). Because the myriad interactions among stressors and biota in the ecosystem cannot be precisely characterized in detail and because all contaminant concentrations and biota populations and exposures cannot be known throughout the floodplain, there are sources of uncertainty inherent in the risk assessment process. Therefore, the results of the ERA must be seen as approximations that may not be precisely correct but are nevertheless sufficiently reliable to guide remedial actions that are intended to be protective of the ecosystem.

Uncertainties inherent in each of the major risk assessment activities are addressed in the following discussion. Whenever possible, a direction (under or overestimate) has been given to the type of uncertainty or its effect on the results of the ERA.

6.4.3.1 Problem formulation

Assessment and measurement endpoints were chosen to indicate whether indicator components of the EFPC floodplain ecosystem are at risk from various stressors, including chemical and non-chemical stressors (Sect. 6.1.3). Along with the environmental description, which defines the exposure setting in the EFPC floodplain ecosystem (Sect. 6.1.4), these endpoints focused the design and activities of the ecological investigation and the subsequent risk characterization.

Indicator organisms. A limited set of indicator organisms was used to infer risks to organisms in various trophic levels including predators. Although these indicators do not constitute the majority of species in either aquatic or terrestrial food chains, they should be representative of key species that would be exposed to contaminated physical media in the aquatic and terrestrial ecosystems and should indicate the potential for exposures to predators. Biomagnification, the accumulation of higher contaminant body burdens with increasing trophic distance from the contaminant source, was observed when small carnivorous animals (shrews and

wrens) were compared to terrestrial insects. Body burdens in shrews and wrens, rather than the less contaminated mice and vole, were used to evaluate potential exposures of carnivores. This was adjusted in part with the feeding range/dietary analysis. Therefore, it is unlikely that projected risks to larger carnivores was underestimated.

Sample sites. Limitations of time and money prevented extensive sampling and analysis of biota. The biological survey and sampling areas comprised a small area out of a much larger area in the EFPC floodplain and, therefore, may not be representative of the entire EFPC floodplain. To make best use of the limited sampling and analytical resources, sample sites were chosen to evaluate the effects of releases close to the Y-12 Plant (Site 1), high levels of contaminants in the soil (Sites 2 and 3), and contributions of the Oak Ridge Sewage Treatment Plant (Site 4) and Bear Creek (Sites 5 and 6). Reference areas previously used for BMAP were used in this study so historical data from the reference sites could be considered along with reference data obtained in this study. Per the study design, the location of sample sites is more likely to have caused an overestimate of exposures than an underestimate.

Timing of sampling. A single round of surveys and sample analysis was used in the EFPC ERA. Because many environmental processes are seasonal, phenomena that would be present at other seasons (ephemeral spring plants, nesting birds, aquatic brood stock) may not have been observed. Therefore, absence or low numbers of seasonally variable organisms may not be significant in population surveys, and attributing such observations to site contaminants would be an overestimate of impacts. Immature organisms that had not equilibrated with environmental contaminants may have been sampled. The degree of equilibration with the exposure environment was not evaluated, so the observed body burdens may have underestimated exposure of the reproductive population.

6.4.3.2 Characterization of exposure

Uncertainties in exposure characterization arise mainly from uncertainties inherent in study design and analytical limitations.

Aquatic sampling site selection. Evaluation of contaminant concentrations in surface water samples shows that the Y-12 Plant is the primary source of water-borne contaminants. Aquatic biota sampling sites included one site close to the Y-12 Plant, so it is unlikely that the maximum exposure of aquatic biota to water-borne contaminants was underestimated. An evaluation of the nature and extent of sediment contaminants was done, also. Regardless, exposure of aquatic biota to sediment contaminants may have been underestimated.

Domination by the Y-12 Plant source. Impacts to aquatic biota in EFPC from the release of water-borne contaminants from the Y-12 Plant may mask any effects that occur to these biota due to other factors such as input of contaminated floodplain soils, urban runoff, or effluent from the Oak Ridge Sewage Treatment Plant. Although the effects of these potential contaminant sources can best be evaluated after the release of contaminants from the Y-12 Plant is stopped, attributing all of the observed exposures in aquatic biota to releases from the Y-12 Plant overestimates the Y-12 Plant impact, while attributing the aquatic exposures to non-Y-12 Plant sources would greatly overestimate the impact of EFPC floodplain sources.

Contaminant concentrations in physical media. Some aquatic contaminants are toxic at concentrations below detection limits. For example, mercury and Aroclor 1260 concentrations in surface water and sediments below the detection limit might nevertheless exceed concentrations capable of producing toxic effects to aquatic species. Therefore, reliance on the identification of contaminants detected in surface water can underestimate risks to aquatic biota.

Terrestrial sampling site selection. Sample and survey sites for terrestrial biota included areas with the highest observed soil concentrations of mercury and PCBs. Therefore, site selection was unlikely to lead to underestimates of the maximum exposures of terrestrial biota. Because the study design was biased toward locations expected to be contaminated, terrestrial exposures may be overestimated by the study design. However, calculations of maximum exposure are intended to show only whether a risk exists in the study area. Exposure data are to be used in the feasibility study (FS) for the selection of preliminary remedial goals (PRGs), which are applied on a site-specific basis to delineate areas requiring remediation. Site-specific application of PRGs reduces system-wide overestimates of risk that result from extrapolating exposures at highly contaminated areas to the entire system.

Selection of COCs. COCs were selected following EPA's data evaluation criteria (EPA 1989c). These criteria are intended to be conservative; they allow contaminants that are not of concern to be screened out to reduce the set of contaminants that must be evaluated. Risk characterization must subsequently evaluate the importance of each COC. Therefore, the selection of COCs overestimates the range of harmful contaminants, but it does not overestimate risk.

Background data. Some indicator organisms were not found at the reference site. The absence of reference data prevents a comparison of site-specific exposure to background. Therefore, some site-specific exposures that would have been discounted by comparison to background data may be overestimated. However, contaminant body burdens at the reference

site are expected to be low, so the effects of overestimates resulting from missing background samples on the overall exposure evaluation should be small.

Proportion of contaminant body burden attributable to food. Mercury and Aroclor 1260 concentrations in the diets of the indicator organisms are not known, but diet probably contributes the major fraction of the observed body burdens. Contaminant concentrations in the diet of aquatic organisms are ultimately related to the surface water and/or sediment contaminant concentrations, whereas contaminants in the diets of terrestrial organisms may be related to soil contaminant levels by uptake into plants or soil-dwelling prey or in water through ingestion of aquatic prey.

Uncertainty in risk to terrestrial animals includes uncertainty about their feeding range. The extent of risk to predators depends on the relative contribution of contaminated prey to the predators' diets. Because sources of contaminants are limited to the EFPC floodplain, the risk to predators depends on their foraging habits. The fraction of a top predator's diet that comes from the contaminated portion of the EFPC floodplain is variable, but typically should be $< 10\%$ for foxes or raptors. The average quotients for protection of predators from consumption of mercury in mice were low, but for shrews and wrens the quotients were higher. Predators that feed mostly in the EFPC floodplain and whose diet consists of a preponderance of small carnivorous prey would be at risk for toxic effects of mercury, while those that range farther and have a more varied diet may not be at significant risk.

EFPC is the dominant source of aquatic prey in the East Fork Valley. Therefore, uncertainties in the exposure of predators consuming aquatic prey are less if the hunting range of the predators is restricted to the EFPC floodplain. Because the overall productivity of EFPC increases and the body burdens of most contaminants decrease with distance downstream, exposure of predators to contaminants in aquatic prey would likely be overestimated by using averages of the observed body burdens. In addition, there is uncertainty about the feeding range of piscivorous birds such as herons, which may be exposed to contaminants outside the EFPC floodplain. Attributing contaminant body burdens in herons to EFPC may be an overestimate of their exposure to EFPC contaminants.

Accuracy of analyses. According to EPA CLP guidelines, recovery limits for surrogates of semivolatile organic analytes may range from 10% to 141% in water and from 18% to 137% in soil; recoveries for the pesticide surrogate can range from 25% to 150%. Contract-required surrogate recoveries from biological tissues in this study were 31% to 142% for organic analytes and 75% to 125% for inorganic analytes. An evaluation of data quality presented in Sect.

6.1.5.4 shows that analytical results for biota met the analytical objectives. However, the accuracy of contaminant concentrations used in calculations is variable. Because of inherent limitations in extraction of organic analytes, results of organic analyses may underestimate true concentrations by a factor of 2 or 3. Analyses of inorganic analytes were judged to be within 25% of the true value. No corrections were made in this study for variability in surrogate recovery.

6.4.3.3 Characterization of ecological effects

The characterization of ecological effects includes evaluation of experimental and media toxicity data and biological surveys.

Toxicity values. Criteria used to calculate risk quotients were derived from a limited set of experimental and observational data. Body burden data were used as evidence of exposure and, when linked to effects, as effects too. In most cases, they were extrapolated from results obtained with test organisms that were different from EFPC indicator organisms being evaluated. Whenever possible, criteria were the lowest observed effects level in the test organism, which likely overestimates effects on populations. Therefore, conclusions based on these quotients should be considered overestimates of risk and advisory rather than definitive.

Variability in population survey data. Except for birds, insects, and small mammals, population surveys consisted of a single sampling event at each site, so the measures of central tendency variability could not be determined. Thus, parametric statistical comparisons could not be performed on the population data. The analysis of the population data was done by a weight-of-evidence analysis, which took into account longitudinal trends, correlations of effects, and temporal trends from BMAP. The nature of these uncertainties is not defined, but because causality was not attributed unless the results were scientifically convincing, actual risks may have been overestimated.

6.4.3.4 Risk characterization

Risk characterization integrates the effects of exposures to stressors and stressor responses of the indicator organisms. Therefore, all of the uncertainties inherent in problem formulation, exposure characterization, and characterization of ecological effects are combined. In addition, there are uncertainties in extrapolating current exposures to the future.

Cumulative risks. Risks are typically estimated for single contaminants, single exposure pathways, and single indicator species. Cumulative risks can be estimated by summing the individual risk estimates. Generally, the methods used in risk characterization are sufficiently

conservative that individual risks are overestimated. However, deleterious effects in ecosystems may cascade throughout the system. For example, a contaminant-related reduction in the ability of small mammals to reproduce may cause a food shortage to predators that are not directly affected by the contaminant. Therefore, summed risks in aquatic and terrestrial food chains may underestimate actual risks to biotic receptors.

Future exposures. Both the distribution of aquatic and terrestrial habitats and the transport of contaminants from sources to receptors may be altered by changes in land use or in operations at the Y-12 Plant. No clear causal relation among contaminant distribution, biotic effects, and habitat type in the EFPC floodplain was observed in this investigation. Therefore, the effects of exposure on habitat or ecological succession must be evaluated conservatively. Changes in operations at the Y-12 Plant may have marked effects on the flow regime of EFPC, altering levels of chemical and physical stressors and the transport of contaminants into and out of the EFPC floodplain. Past construction and remediation have been associated with increased contaminant body burdens in aquatic biota in lower EFPC (Loar 1992; Hinzman 1992). The concentrations and timing of such releases associated with decommissioning and decontamination activities at the Y-12 Plant are uncertain.

6.4.3.5 Summary

Sources of uncertainty in the EFPC ERA are indicated in Table 6.85. Major sources of uncertainty in problem formulation are the characterization of the transport of contaminants from soil, sediment, and water through the food chain and in the temporal limitations of the environmental characterization. Both factors may lead to underestimates of risk. The largest sources of uncertainty in exposure assessment are analytical limitations that result in the inability to detect contaminants that may be bioaccumulated or biomagnified, and a lack of exact knowledge about the foraging behavior of predators in EFPC. The former may lead to an underestimate of exposure, whereas the latter may lead to an overestimate of exposure because overly conservative assumptions about food sources may be made. The major sources of uncertainty in effects assessment are extrapolation of conventional toxicity data to the EFPC indicator organisms and the small number of samples taken during biological surveys to provide knowledge of field-observed effects. Because they are overly conservative, both factors may lead to an overestimate of risks.

The ecological risk characterization probably leads to an overestimate of individual risks to taxa in the EFPC floodplain ecosystem. That overestimate is intended to protect the animals and plants in the ecosystem and is, therefore, acceptable. However, cumulative risks include

Table 6.85. Summary of uncertainties in the ERA

Risk parameter	Underestimate	Overestimate
Problem formulation		
Indicator organisms	+	
Sample sites		+
Timing of sampling	+	
Exposure characterization		
Surface water exposure	+	
Y-12 source	+	
Terrestrial exposure		+
COC selection		+
Background data		+
Foraging habits		+
Analytical accuracy	+	
Effects characterization		
Toxicity criteria		+
Variability in survey data		+
Risk characterization		
Cumulative risk		
Aquatic food chain	+	
Terrestrial food chain	+	
Future risk		
Aquatic food chain	+	
Terrestrial food chain	+	

antogonistic/synergistic effects and unforeseen consequences in the food chain and, therefore, are probably underestimated. Because potential releases from the Y-12 Plant may increase as a result of future cleanup activities, extrapolation from current conditions is especially likely to underestimate future risks.

6.4.4 Comparison to Human Health Risks

6.4.4.1 Current impact

The human health risk assessment concludes that there is no immediate threat to human health associated with contaminants in the EFPC floodplain. Similarly, no immediate threats to threatened and endangered species or to terrestrial flora were demonstrated in the ERA. Weight-of-evidence arguments showed that aquatic populations are currently impacted by water-borne contaminants that result in elevated contaminant body burdens and changes of community structure, including the absence of species in habitats where they are expected to occur. Further, there is evidence from BMAP that the aquatic community is recovering slowly, but is not recovered. Terrestrial fauna are experiencing various levels of effects and risk. These ecological effects are important negative impacts that justify remedial actions.

6.4.4.2 Risk criteria

In both risk assessments, risk estimates exceeded criteria established for acceptable risk. That is, in the human health risk assessment, hazard indices exceeded 1 for a number of COCs and exposure scenarios. In the ERA, criteria described in the assessment endpoints were exceeded.

6.4.4.3 Contaminant sources

Contaminated soil and groundwater were the most important contaminant sources in the human health risk assessment, whereas contaminated soil was the major source to terrestrial flora and fauna. In contrast, water-borne contaminants released from the Y-12 Plant were found to be the most important source of contaminants to aquatic biota.

6.4.4.4 Exposure pathways

Initial calculations showed soil ingestion, groundwater ingestion, and exposure through the food chain via garden vegetables to be the most important exposure pathways to humans. However, consideration of uncertainties in contaminant transport through the food chain and in groundwater use resulted in the conclusion that these pathways are probably not of major importance to human health. Therefore, soil ingestion is the most significant pathway of human

exposure. In contrast, the food chain appears to be the primary exposure pathway for both aquatic and terrestrial fauna. Direct contact with surface water is also an exposure pathway for aquatic biota. Direct contact with and ingestion of soil are probably significant to the health of terrestrial biota only as points of entry into the food chain, in which predators ingest contaminants taken up by their prey from soil and possibly surface water.

6.4.4.5 Contaminants of concern

The most important COCs identified in the human health risk assessment were mercury in soil and arsenic in groundwater. Volatile and semivolatile organics, pesticides, PCBs, and radionuclides were not shown to be of major concern for human health. Both mercury and PCBs were shown to be dominant COCs in aquatic and terrestrial biota. PCB was a major contaminant in every taxon analyzed, and mercury was found in all but the vole. The potential impact of other inorganic contaminants appears to be minor. PAHs and pesticides were shown to be of little concern in the ecosystem.

6.4.4.6 Spatial distribution

Because the human health risk assessment concludes that ingestion of mercury in contaminated soil in a residential or agricultural scenario constitutes the most important human health risk, the spatial distribution of risk follows the distribution of mercury in soil in residential areas of the EFPC floodplain. Potential future exposures were judged to be unacceptable in every spatial risk assessment segment of EFPC floodplain. Current human health risks in the EFPC floodplain were judged to be greatest in the agricultural setting from 44000 E to 49000 E and in the residential setting in the segments approximately located from 40000 E to 44000 E, from 49000 E to 50700 E, and from 56000 E to 58800 E. Exposures to terrestrial plants are also related to soil contaminant concentrations, but it is not limited to residential areas. Therefore, unacceptable exposures may also occur in the highly contaminated area near EFK 22 (~33000 N to 34500 N). Risks to aquatic biota are generally greatest immediately downstream from the Y-12 Plant (EFK 22-23, ~31000 N to 34500 N), and with some species-specific exceptions, are not related to the distribution of contaminants in EFPC floodplain soils. Terrestrial body burdens are generally highest at the three upstream sampling sites and are not clearly related to the distribution of contaminants in soil.

6.4.4.7 Conclusions

The human health risk assessment indicates a potential need for remediation of EFPC floodplain soils in residential or potentially residential areas. In contrast, the ERA shows that

the most important potential remedial action to mitigate observed impacts on the ecosystem is protection of aquatic communities by preventing releases of water-borne contaminants into EFPC by the Y-12 Plant. Table 6.86 summarizes the discussion above. Additionally, remediation of selective soils would reduce risk to terrestrial organisms. Data are available to determine what areas of the EFPC floodplain may need to be remediated to protect terrestrial habitats and communities. These evaluations will be done during the FS.

6.4.5 Summary of Ecological Risk Characterization

Under current conditions, aquatic biota in EFPC are subject to exposures exceeding criteria for their protection. Table 6.87 summarizes the ranges of risk quotients derived by dividing concentrations of contaminants in exposed environmental resources by various protective criteria values. In general, higher quotients tend to cluster at the upstream sampling sites.

Changes in taxonomic richness and diversity, taxonomic composition of populations, and contaminant body burdens in aquatic biota with distance downstream from the Y-12 Plant imply that waterborne contaminants being released from the plant are the major causes of current risk to the aquatic biota. Weight-of-evidence arguments support the propositions that waterborne releases of mercury and PCBs account for the high body burden concentrations of these two contaminants in fish and benthos in EFPC and produce toxic effects to fish and benthic communities in EFPC, resulting in communities with fewer total taxa, lower diversity, and a predominance of taxa that are tolerant of degraded water quality conditions.

The distribution of habitats in EFPC and its floodplain is not indicative of acute chemical or physical stress. Numbers of birds, insects, and small mammals observed in the EFPC floodplain were not depressed compared to the reference survey areas. However, contaminant body burdens in aquatic biota, insects, small mammals, and birds exceed criteria for protection of predators (Table 6.88), suggesting that raptors and mammalian predators that hunt predominantly in the EFPC floodplain, including part of the reference site, may be at risk through accumulation of toxic amounts of mercury. High risk quotients for terrestrial biota are distributed throughout the EFPC floodplain with the highest risk in the upper part of the floodplain.

6.5 SUMMARY AND CONCLUSIONS

The FFA among DOE, EPA, and TDEC for the EFPC site requires a comprehensive investigation to determine the nature and extent of contamination in the floodplain. Under

Table 6.86. Comparison of human health and ecological risks[a]

Risk assessment endpoint	Human health risk	Ecological risk	
		Aquatic	Terrestrial
Risk criteria exceeded	+	+	+
Contaminant source			
Y-12 Plant	-	+	-
EFPC floodplain soil	+	-	+
Exposure pathway			
Soil ingestion	+	-	-
Food chain	(+)[b]	+	+
Groundwater ingestion	(+)[b]	-	-
Surface water	-	+	-
Primary COCs			
Inorganic			
Mercury	+	+	+
Arsenic	+	-	-
Cadmium	+	-	-
Selenium	-	+	+
Zinc	+	-	-
PAHs	-	-	-
Pesticides	-	-	-
PCBs	-	+	+
Radionuclides	-	-	-

[a] - = endpoint not met
+ = endpoint met or exceeded
[b] RME calculations indicate risk, but uncertainty analysis discounts significant risk.

Table 6.87. Classification of risk quotients for aquatic exposures by range and location[a]

Sample site	Quotient range[b]				
	Q>100	10≤Q<100	1≤Q<10	0.1≤Q<1	Q<0.1
1		WQC, Hg AI, pred. SR, PCB tox. SR, PCB pred.	CR, Hg tox. CR, PCB pred. RS, PCB tox. CR, PCB tox. RS, PCB pred.	WQC, Zn SR, Hg tox. RS, Hg tox.	
2		WQC, Hg SR, PCB tox. SR, PCB pred.	SR, Hg tox. WR, Hg tox. AI, pred. RS, PCB tox. RS, PCB pred.	WQC, Zn RS, Hg tox. CR, Hg tox. CR, PCB tox. CR, PCB pred.	
-3		WQC, Hg	AI, pred. SR, PCB tox. RS, PCB tox. SR, PCB pred. RS, PCB pred.	WQC, Zn SR, Hg tox. RS, Hg tox. CR, Hg tox. CR, PCB tox. CR, PCB pred.	
4			AI, pred. SR, PCB tox. RS, PCB tox. SR, PCB pred. RS, PCB pred.	WQC, Zn SR, Hg tox. RS, Hg tox.	
5	HN, pred.	AI, pred.	HN, Hg tox. SR, PCB tox. RS, PCB tox. SR, PCB pred. RS, PCB pred.	WQC, Zn SR, Hg tox. RS, Hg tox. HN, PCB tox.	
6			SR, PCB tox. SR, PCB pred.	WQC, Zn CR, PCB tox. CR, PCB pred.	SR, Hg tox. RS, Hg tox. CR, Hg tox. CR, pred.

Table 6.87 (continued)

Sample site	Quotient range[b]				
	Q>100	10≤Q<100	1≤Q<10	0.1≤Q<1	Q<0.1
Reference				WQC, Zn SR, Hg pred. RS, Hg pred. CR, Hg pred. CR, Hg pred. AI, Hg pred.	SR, Hg tox. RS, Hg tox. CR, Hg tox. SR, PCB tox. RS, PCB tox. CR, PCB tox. SR, PCB pred. RS, PCB pred. CR, PCB pred.

[a]Key:
WQC = Water quality criteria
SR = Stoneroller
RS = Redbreast sunfish
CR = Crayfish
AI = Aquatic insect
tox. = toxicity to indicator organism
pred. = toxicity to predators
HN = Heron

[b] Quotients based on concentrations below detection limits were not included.

Table 6.88. Classification of risk quotients for terrestrial exposures by range and location[a]

Sample site	Quotient range[b]				
	Q>100	10≤Q<100	1≤Q<10	0.1≤Q<1	Q<0.1
1	EW, pred.			TI, PCB pred. MS, PCB pred.	TI, PCB tox. MS, PCB tox.
2	EW, pred.	MS, pred. WR, pred.	WR, Hg tox. TI, pred.	MS, Hg tox. TI, PCB pred.	TI, PCB tox. MS, PCB tox. WR, PCB tox. MS, PCB pred. WR, PCB pred.
3	EW, pred.	TI, pred.		TI, PCB pred.	TI, PCB tox. MS, PCB tox. MS, PCB pred.
4	EW, pred.	SH, pred. WR, pred.	SH, Hg tox. WR, Hg tox.	WR, PCB tox. MS, PCB pred. SH, PCB pred. WP, PCB, pred.	TI, PCB tox. PCB tox. SH, PCB tox. PCB pred.
5	EW, pred. SH, pred.		SH, Hg tox.	SH, PCB pred. HN, PCB pred.	TI, PCB tox. MS, PCB tox. SH, PCB tox. TI, PCB pred. MS, PCB pred.
6		EW, pred.			TI, PCB tox. TI, PCB pred.
Reference			MS, Hg pred. EW, Hg pred.	MS, Hg tox.	TI, Hg tox. MS, HG tox. TI, PCB pred. MS, PCB pred.

[a] Key:
EW = Earthworm
TI = Terrestrial insect
MS = Mouse
SH = Shrew
WR = Wren
tox. = toxicity to indicator organism
pred. = toxicity to predators

[b] Quotients based on concentrations below detection limits were not included.

CERCLA, prior to site remediation, the RI/FS process must be executed, which includes conducting a baseline ecological risk assessment.

The baseline ecological risk assessment characterizes baseline risk levels, helps determine the need for remedial action, provides a basis for deciding what levels of contaminants can remain on the site, and provides a basis for comparing remedial alternatives. In addition, the work evaluates if and where there is imminent and substantial danger to animal and plant populations and their habitats. The EFPC ERA, which is structured according to the *Framework for Ecological Risk Assessment* (EPA 1992a), consists of four interrelated activities:

(1) Problem Formulation — establishes the goals, breadth, and focus of the assessment; provides a preliminary characterization of chemical contaminants and physical stressors present in the ecosystem, the components, especially indicator organisms, of the ecosystem likely to be at risk along with assessment and measurement endpoints; also, the ecological contaminants of concern are defined.

(2) Exposure Characterization — evaluates the interactions of the stressors with the ecosystem attributes; describes the biotic and abiotic ecosystem attributes, along with the route, magnitude, frequency, duration, trend, and spatial pattern of exposure of each indicator population or habitat component in relation to chemical contaminants; emphasis is placed on body burdens of indicator organisms for evaluation of chemical exposures.

(3) Effects Characterization — evaluates the ecological response to chemical contaminants and physical stressors in terms of the selected assessment and measurement endpoints, and, depending on the parameters of exposure, results in a profile of response to contaminants at concentrations to which populations and habitats are exposed; data from both field observations and controlled laboratory studies are used to evaluate ecological effects.

(4) Risk Characterization — aggregates the effects of exposure and stressor response on indicator populations or habitat components, using risk quotients (a ratio of exposure value to effect value) with statements of uncertainty and then interprets these data using the weight-of-evidence approach for defining cause/effect relationships that can be expressed as risk; both current and future conditions are discussed.

More specific objectives of the sampling and analysis of indicator populations and their associated habitats and environmental media were advanced in Sect. 6.1.

The problem was formulated in terms of assessment endpoints, which are statements of ecological values that, if found to be significantly affected, would indicate the need for remediation. Assessment endpoints are the focus of remedial decisions. Assessment endpoints were selected to meet the requirements of the various environmental trustee agencies within the context of goals. For example, the Migratory Bird Treaty Act, CERCLA, and NEPA provide protection to migratory birds. Therefore, one assessment endpoint addresses population impacts to migratory birds. The other endpoints are provided in Sect. 6.1 and also in Table 6.89, along with a short statement of their results.

As documented in the problem formulations phase of this ERA (Sect. 6.1.3), 11 assessment endpoints were identified that would help to determine whether existing site conditions provided attainment of basic policy goals for the EFPC floodplain. Following the conclusion of the exposure characterization, effect characterization, and risk characterization phases, and within the limits of the uncertainties associated with this type of study, the risk assessment can be compared to these baseline assessment endpoints. Table 6.89 provides a listing of the defined endpoints along with the respective ERA results from direct surveys or risks characterization analyses (quotient and weight-of-evidence). A few of the key results and conclusions of this comparison are:

(1) a few (assessment endpoint 1 and possibly 2, 4, and 9 in Table 6-89) of the assessment endpoints were achieved with existing EFPC contaminant conditions; and

(2) measurements or risk characterization method results indicate that the rest of the assessment endpoints can not be met with existing EFPC floodplain conditions, i.e., many body burdens exceed the protective concentrations.

In the EFPC ERA, ecological assessment endpoints are supported by numerous ecological and physical media measurements or measurement endpoints. Indicator organisms were selected to represent organisms in various habitats to determine whether those groups of organisms are or have potential to be negatively affected by contamination at a site. Measurement endpoints included population size and community structure, contaminant distributions in physical media, and contaminant body burdens in indicator biota. Both lowest trophic level and susceptible indicator organisms were measured. This process provides an overall picture of the plant and animal populations and habitats and provides the decision-maker with a comprehensive view of not only the existing risk but also the information needed to eventually select remediation alternatives. The following narrative provides an understanding of the terrestrial and aquatic habitats and the influences of chemical and physical stressors on the plant/animal communities.

Table 6.89. Comparison of ERA results with assessment endpoints

Assessment endpoint	ERA result
1. No harm to any threatened and endangered species and their critical habitat of the EFPC floodplain	1. Literature and field surveys did not reveal threatened and endangered species living in EFPC floodplain. Habitat that could support an expansion of the range of a threatened and endangered species (e.g., Indiana and Gray bats) was found. However, a range expansion of such species is not likely.
2. Maintenance of plant community composition and/or structure required for rare plant and animal and support species	2. Literature and field surveys did not find that potential habitat—plant community—for rare animal or support species was harmed by EFPC contaminants. Wetlands may be an exception depending on the preliminary remediation goal.
3. No killing or harming of migratory birds as a result of exposure to site-specific stressors	3. Field survey evidence provides names of more than 30 migrant bird species which are likely ingesting contaminated worms and arthropods and, therefore, likely being affected by EFPC contaminants.
4. The presence and structure/function of wetlands in relation to contaminants	4. Seventeen wetlands exist in the floodplain, some with mercury contamination, but each appears to be functioning.
5. Fish communities in which the proportion of species tolerant of degraded water quality is $<30\%$	5. Continual releases of water-borne contaminants impact fish community structure, resulting in communities dominated by species tolerant of degraded water quality conditions at the 4 sites closest to the Y-12 Plant.
6. Ratio of contaminant concentration in surface water to water quality criteria for protection of aquatic life ≤ 1	6. The quotient method indicated significant exceedances by factors up to 45 from mercury concentrations above water quality criteria of EFPC below Lake Reality.
7. Aquatic indicator organisms contaminated body burden ratio to toxicological effects levels ≤ 1	7. Contaminant body burden measurements and quotients of 1 or less for mercury and up to 20 for PCBs were determined for stonerollers, redbreast sunfish, and crayfish.
8. Fish contaminant body burden ratio to protect piscivorous biota levels ≤ 1	8. The quotient method identified numerous exceedances by factors of 10 to >100 for mercury and up to 16 for PCBs for fish contaminant body burdens in a predator pathway.
9. Terrestrial animals with diversity, abundance, and distributions indicative of undegraded conditions resulting in ≥ 20 or more percent decrease compared to the reference.	9. Some organisms missing from certain sampling sites may indicate large decreases in abundances. Other findings suggest that no 20% decreases occur.
10. Ratio of contaminant body burdens in terrestrial indicator species to toxicological effects levels ≤ 1	10. Body burdens of heron, wrens, and shrews were found to exhibit quotient ranges between about 2 and 8 for mercury in the ERA sampling.
11. Ratio of contaminant body burdens in terrestrial indicator species to levels protective of terrestrial predators ≤ 1	11. Contaminant body burden quotients in excess of 100 were determined for herons, earthworms, and shrews for predator pathways. Quotients for mercury were as high as 19 for mice and 70 for wren predators.

The terrestrial portion of the EFPC study area is characterized by cleared areas, shrubs, herbaceous plants, and second-growth trees that form thick stands up to the creek banks in many locations. Floodplain soils are typical silty loams. The forests are dominated by mixed hardwood species that are tolerant to flooding. Even in the vicinity of developed areas, thick stands of trees typically abut the creek. Because of different land uses in the past, the woods are in various stages of ecological succession. A variety of shrubs and herbaceous species are also present; these flourish in areas where the over-story has been removed and more light is available. Wetlands are also found in the floodplain. Portions of the floodplain have been filled and developed commercially, and in places the creek has been channelized so that it has little or no natural edge. Mid-reaches of the floodplain contain several large agricultural tracts. Other portions of the floodplain consist of grass and old field habitats. The location and extent of terrestrial habitats have been documented and are retrievable on a geographical information system (GIS). In all of these terrestrial habitats, animals, including deer, raccoon, and birds (residents and migrants), are evident.

The upper reaches of EFPC aquatic habitats average ~4 m (160 in.) width and 0.25 m (10 in.) in depth, increasing to an average width of ~10 m (400 in.)and a depth of >0.5 m (20 in.) where the creek reenters ORR, with deeper pools at some locations. Aquatic habitats consist of a mix of riffles, runs, and pools whose locations have been entered on a GIS. The banks are characterized by unconsolidated sediments, and the bottom varies from exposed rock or limestone rip-rap to smooth, muddy sediment. The stream contains fish of various species as well as benthic and other organisms.

Environmental risks come from two distinct sources. The preponderance of risk to aquatic biota in the upper part of the watershed below Lake Reality comes from waterborne contaminants being released from the Y-12 Plant during current operations. Contributions by leaching of contaminants from EFPC floodplain soils and sediments are masked by these ongoing releases. Exposure to contaminants in the aquatic environment has caused a significant degradation of stream quality. In contrast, exposures to terrestrial biota come from historical contaminant deposits in the EFPC floodplain. Some of the historical depositions of mercury and other contaminants are buried under less contaminated depositions of sediment from flooding. The soil exposures provide for significant risk to predators of earthworms and insects. Exposures via these prey to small mammals and birds may be high. There is less risk to top predators that obtain a portion of their food outside the EFPC floodplain.

Surface water is the primary medium of exposure for aquatic organisms to contaminants currently being released to EFPC via surface water from the Y-12 Plant and to contaminants in

floodplain soils that are transported via erosion and surface runoff. Soil is the primary medium of exposure for terrestrial biota. Stream sediments are a second major exposure route for benthic invertebrates and for terrestrial consumers by way of flood deposits on vegetation and in wetlands. In both aquatic and terrestrial communities, indirect exposure of organisms via the food web is important. Methylation/demethylation of mercury in sediments and stream water and biodegradation of organic contaminants are the most important chemical transformations.

Past studies have found elevated levels of mercury and PCBs in fish, with the highest concentrations generally in the upper reaches of EFPC near the Y-12 Plant. PAHs were concentrated by clams placed in EFPC. Historical sampling has detected mercury in or deposited on pasture grass on the EFPC floodplain, with low but detectable concentrations in deer. Mercury concentrations in EFPC physical media are considerably greater than background concentrations for all media sampled. A total of 13 aquatic species from EFPC had total mercury concentrations exceeding the background level, and mercury was particularly elevated in six fish species, frogs, snapping turtles, and crayfish. Other priority pollutant metals that were elevated are arsenic (crayfish), beryllium (fish), cadmium (fish, crayfish, frogs), chromium (fish, crayfish, frogs, turtles), copper (fish), selenium (fish), silver (fish, crayfish, turtles), and thallium (fish). Among the priority pollutant organic compounds, PCBs were significantly elevated in fish.

Overall, both historical and current studies of bioaccumulation showed generally decreased body burdens of mercury and/or PCBs/pesticides in stonerollers, sunfish, crayfish, earthworms, and certain terrestrial insects with increasing distance downstream from the Y-12 Plant, and higher body burdens than in organisms from presumably uncontaminated reference sites. BMAP studies have shown that fillet concentrations for mercury and PCBs have decreased since the late 1980s, possibly as a result of prior remediation activities. Recently, mercury concentrations in redbreast sunfish have decreased near the Y-12 Plant, and peak values in redbreast sunfish were noted at Site 3, 6.4 km (3.8 mi) downstream from Lake Reality. The body burden of PCBs in sunfish was approximately constant through Site 3 and decreased at sites downstream. Thus, there is evidence of recovery, but the stream is not recovered.

Toxicity tests on a variety of aquatic and terrestrial organisms using EFPC surface waters, soils, or sediments have shown deleterious effects but have failed to provide conclusive evidence of consistent spatial patterns. *Ceriodaphnia* showed undepressed survival rates in 7-d tests at sites near the Y-12 Plant, and fingernail clams showed reduced survival and reproduction at the EFPC sites nearest the Y-12 Plant and in EFPC compared with reference sites.

Under current conditions, aquatic biota in EFPC are subject to exposures exceeding criteria for their protection. In general, higher risk quotients tend to cluster at the upstream sampling sites, indicating that waterborne contaminants released from the Y-12 Plant are the major source of exposure to aquatic biota. The number of fish species, benthic macroinvertebrate families, and insect adult individuals at EFPC generally increased with increasing distance downstream from the Y-12 Plant. Compared with a reference site, EFPC had fewer species of fish, fewer families of benthic macroinvertebrates, and lower mean numbers of individuals per sample of adult insects of terrestrial origin. The number of fish species classified as tolerant of degraded conditions decreased downstream in EFPC and was higher at four of the six sites in EFPC than at the reference site. Taxonomic diversity of fish, benthic macroinvertebrates, and adult insects increased downstream, except at Site 3, and were lowest at the 3 sites where surface water and soil mercury concentrations were greatest. Taxonomic diversity for fish, benthic macroinvertebrates (families), and adult insects with aquatic larval forms at EFPC sites were generally less than at the reference site. The richness of families from sensitive arthropod orders of Ephemeroptera, Plecoptera, and Trichoptera was lowest at the EFPC site nearest the Y-12 Plant and was lower at all EFPC sites than at the reference site. Studies of periphyton colonization and growth showed less obvious patterns.

The only evidence of effects to terrestrial vegetation in areas with high soil mercury concentrations was low recruitment of the dominant tree species at Site 3. The observed concentrations of contaminants in physical media and biota were not sufficiently high to pose a risk of acute toxicity by short-term contact or consumption. Rather, effects and risk arise from chronic exposures. No threatened or endangered species whose populations depend on the EFPC floodplain were identified. Habitat exists that could be colonized by such threatened or endangered species as bats, but such colonization is not likely.

Terrestrial predators are at risk through ingestion of earthworms, insects, small mammals, and birds in the EFPC floodplain. The calculated risk to terrestrial predators is distributed throughout the EFPC floodplain. Concentrations of mercury in earthworms and terrestrial insects decrease as a function of increasing distance from Y-12 and with the distribution of mercury in surface soils.

In the absence of remediation, the exposures of EFPC communities to contaminants are not expected to change dramatically. The fish and other aquatic organisms would be expected to maintain contaminant body burdens in relationship to releases from Y-12. Aquatic community structure would continue as described, especially in the upper part of the creek below Lake Reality. Terrestrial organisms will also continue to exhibit contaminant body burdens. Predators

of contaminated aquatic and terrestrial organisms will continue to be at risk, too. If the creek were to erode the buried lenses of mercury, exposure could increase to various indicator organisms.

The goals of remedial activities should include returning the concentrations of contaminants in EFPC surface water and sediment to levels that do not cause the accumulation of mercury and PCBs in aquatic biota to exceed benchmark concentrations for protection of the biota. However, unless water-borne mercury and PCB contamination from the Y-12 Plant is controlled, remedial efforts to reduce these contaminant concentrations in aquatic biota by sediment removal will probably not protect aquatic biota. Remediation of soils does not appear to be necessary for the protection of plant communities. Earthworm and arthropods exhibit high contaminant body burdens. Their predators—shrews and wrens—also exhibit high contaminant body burdens. Small terrestrial animals and birds do not show signs of severe disruption of community structure, but contaminant body burdens in these biota imply that their top predators are at risk. From both aquatic and terrestrial viewpoints there is abundant evidence of various levels of risk to ecological resources. Remediation will be necessary to lower that risk to ecological resources.

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7. DERIVATION OF PRELIMINARY REMEDIATION GOALS

This section presents preliminary remediation goals (PRGs) for contaminants in environmental media of East Fork Poplar Creek (EFPC). These PRGs are based on an evaluation of risks to human health and ecological receptors. An overview of the regulatory use of remediation goals along with methods for deriving these numbers are provided in this section. In addition, an inclusive listing of PRGs for chemicals and radionuclides found in EFPC is provided.

7.1 INTRODUCTION

Response objectives for waste site remediation are overall cleanup objectives established on the basis of the nature and extent of contamination, the resources that are currently or potentially threatened, and the potential for human and ecological exposure. Examples of response objectives include protection of human health and ecological receptors, protection of the groundwater resource, and restoration of contaminated surface soil for current and future use. *Remedial action objectives* are site-specific requirements that define the extent of cleanup required to achieve the response objectives.

Several elements comprise a remedial action objective, including chemical-specific numerical cleanup limits (i.e., *remediation goals* or target cleanup levels) for all affected environmental media, the spatial area of attainment, and the restoration timeframe. The U.S. Environmental Protection Agency (EPA) has specified two "threshold criteria" as the basis for deriving target cleanup levels for contaminated environmental media at waste sites (EPA 1988a, 1989a). These criteria are:

- concentrations of chemicals in the environment must comply with applicable or relevant and appropriate federal and state requirements (ARARs), and

- the remediation objectives must afford overall protection of human health and the environment.

EPA has stated that these "threshold criteria" must be satisfied for a remedial alternative to be eligible for selection (53 FR 51394, 55 FR 8666).

ARARs are not a uniformly derived set of similar standards and do not incorporate consideration of the effects of combined exposure to mixtures of chemicals. As such, ARARs cannot be categorically adopted as target cleanup levels. Although alternatives for site remediation must comply with ARARs, due to site-specific factors (e.g., multiple chemicals and multiple exposure pathways), cleanup levels set at the level of single chemical-specific requirements may not adequately protect human health or the environment. If an ARAR is not protective, does not exist for the specific chemical or pathways of concern, or multiple contaminants result in an unacceptable cumulative risk, remediation objectives are developed through the risk assessment process. "Health advisory" levels should be identified or developed to ensure that a remedy is protective.

For carcinogenic effects, these health advisory or risk-based levels are to be selected such that the total (excess) risk of all contaminants falls within the acceptable range of 10^{-4} to 10^{-6} (55 *FR* 8666). The 10^{-6} risk level is specified by EPA as a point of departure for determining remediation goals when ARARs are not available or are not sufficiently protective. For noncarcinogenic effects, cleanup levels should be based on acceptable levels of exposure as determined by the EPA reference doses (RfDs), taking into account the effects of other contaminants at the site.

As noted above, the requirement that a remedial alternative meet chemical-specific ARARs does not ensure that the proposed alternative is protective, and thereby potentially acceptable. This can be determined only by: evaluating the combined carcinogenic risk associated with the ARAR limits for all chemicals at a given site (assuming additivity of effect in the absence of data on synergism or antagonism); establishing that ARARs do not exceed EPA RfDs for noncarcinogenic effects, and are sufficiently protective when multiple chemicals are present; determining whether ecological effects (in addition to human health considerations) are adequately addressed by the ARARs; and evaluating whether the ARARs adequately cover all significant pathways of human exposure identified in the baseline risk assessment (BRA).

Establishment of target cleanup levels typically begins during project scoping or concurrent with preliminary remedial investigation (RI) activities. Because these PRGs are established prior to completion of the BRA, they are initially equated with ARARs or other readily available ecological or health-based limits. As the RI and feasibility study (FS) progress, the results of risk assessment and the subsequent identification of additional ARARs modify the PRGs. Ultimately, final remediation goals are derived that ensure that remedial alternatives comply with ARARs *and*

are protective of human health and the environment. The final remediation goals are derived during the FS and are documented in the Record of Decision.

Based on the available EPA guidance, an outline may be developed of the general approach to derivation of remediation goals for the protection of human health as follows:

- identify subject chemicals of concern,
- assemble a listing of all available ARARs,
- identify potential exposure pathways and receptors at risk,
- develop exposure scenarios and characterize environmental concentrations/activities at the points of exposure using available monitoring data and/or the results of environmental fate modeling,
- if ARARs are available for all subject chemicals and environmental media, evaluate the overall protectiveness to human health of exposure to the chemicals at ARAR levels; take into consideration combined exposure across chemicals and multiple pathways,
- if the ARAR levels are found to be protective, adopt these as remediation goals (cleanup levels), and
- if ARARs are not available for all subject chemicals, or are not found to be protective of human health, derive cleanup levels based upon the results of risk assessment.

These steps have been used in developing remediation goals for contaminated environmental media in EFPC.

The exposure pathways that form the basis for risk characterization in the BRA should be used in deriving target cleanup levels. The point to keep in mind is that chemical-specific remediation goals for contaminants must afford overall protection to human health and the environment. Overall protection as defined by EPA (and noted previously) must take into consideration *combined exposure across all chemicals and pathways of concern* for receptor groups at primary risk of exposure.

7.2 DERIVATION OF HEALTH-BASED PRGS FOR EFPC

EPA guidance for deriving PRGs for protection of human health is presented in RAGS Vol. 1, Part B (EPA 1991c). As defined in this document, PRGs are initial cleanup goals that are protective of human health and the environment and comply with ARARs. Ideally, they are to be developed early in the site evaluation process based on readily available information and are subsequently modified to reflect site-specific information and results of the BRA. Final remediation goals are developed as part of the FS process.

The process of deriving a PRG consists of establishing an acceptable target risk value for exposure to a contaminant, and back-calculating the corresponding concentration in the environmental medium or media under evaluation. As discussed in EPA (1991c), the 10^{-6} risk level is specified as the point of departure for determining remediation goals for carcinogens. Therefore, this excess lifetime risk value is used as the target risk level in deriving a PRG for a given chemical under evaluation.

Note the final target risk level may be adjusted on a site-specific basis to a higher or lower level. Factors that are considered in adjusting the risk level include: potential for actual exposure, the ability to detect/monitor the chemicals under evaluation, weight of evidence (uncertainty) of toxicity information, and sensitivities of the population at risk or other non-site related health risks experienced. Technical feasibility and cost effectiveness also may be factors when adjusting the target risk level.

Having established a target risk level, an equation of the following form is used to derive the risk-based preliminary remediation goal for carcinogens:

$$C_i = \frac{TR \times BW \times AT}{CSF \times CR \times EF \times ED} \qquad (1)$$

where

C_i = preliminary remediation goal (medium- and pathway-specific) for chemical i (units of concentration),
TR = target risk level = 10^{-6},
BW = body weight (kg),
AT = averaging time (days),

CSF = cancer slope factor for chemical i $(mg/kg\text{-}day)^{-1}$,
CR = contact (intake) rate (volume/day),
EF = exposure frequency (days/year),
ED = exposure duration (years).

Note that the equation above would be used to derive a cancer-base PRG for a single chemical. The combined risk of exposure to multiple carcinogens via multiple pathways must not exceed 1×10^{-4}.

PRGs also may be derived based upon the potential for adverse noncarcinogenic effects. Note that both noncarcinogenic and carcinogenic effects must be considered in the development of target cleanup levels. Noncancer risk is expressed in terms of the hazard quotient (HQ) for individual chemicals, and a hazard index [(HI): equal to the sum of the HQ values] for combined exposure across chemicals.

HQ is defined as the ratio of intake (or dose) over the EPA-verified RfD. In conducting risk assessment, if the HQ is greater than 1 for any given compound, it is concluded that the potential for adverse noncarcinogenic effects exists. If a receptor is exposed to multiple chemicals, the HQ values for each chemical are summed to derive an HI. A value of 1 also is used to evaluate the significance of the HI.

Equation (2) is used to derive a PRG based on noncarcinogenic effects for a single chemical:

$$C_i = \frac{THQ \times RfD \times BW \times AT}{CR \times EF \times ED} \qquad (2)$$

where

C_i = preliminary remediation goal (medium- and pathway-specific) for chemical i (units of concentration),
THQ = target hazard quotient = 1,
RfD = reference dose (chemical-specific),
BW = body weight (kg),
AT = averaging time (days),
CR = contact (intake) rate (volume/day),

EF = exposure frequency (days/year),
ED = exposure duration (years).

For exposure to multiple chemicals, the variable target hazard quotient would be modified. Instead of a value of 1, the target hazard quotient would be 1 divided by the number of chemicals for which potential exposure was a concern.

To summarize, PRGs have been developed for selected chemicals observed in the Phase Ia and Ib data sets. For each chemical, multiple PRGs have been derived based on sets of relevant exposure pathways (as noted above). In addition, PRGs are derived for two categories of receptors: children ages 3 to 12 years, and adults. Tables 7.1 through 7.8 present the results of preliminary calculations.

The listing of PRGs for human health presented in Tables 7.1 through 7.7 includes all compounds that were evaluated in the Tier II risk assessment (see Sect. 5.). However, remediation will *not* be required for all of these substances. A first attempt was made at this time to be all inclusive and to refine this list as the RI nears completion and the FS is well underway. As an example, the presence of beryllium and manganese in EFPC floodplain soils has not been found to be statistically different from levels in background soils (i.e., at Hinds Creek). Further, the results of the BRA indicated that mercury (noncancer effects) and arsenic (carcinogenic effects) are the principal contaminants driving the human health risk assessment of exposure to soils. Table 7.8 summarizes the human health PRGs for the principal chemicals of concern identified based on the results of the BRA.

For the purposes of comparison, a figure has been prepared depicting the relative magnitude of the PRGs for mercury for soil and sediment pathways. Note in Fig. 7.1 how much lower the PRGs for the produce ingestion pathway are compared to direct soil contact. This directly reflects the conservative exposure assumptions adopted for the foodchain pathways. The extremely high PRG value for sediments reflects the limited exposure potential via this pathway (i.e., limited body surface area exposed and very little transfer across the biological membrane inorganic chemicals).

Table 7.1. Preliminary remediation goals (PRGs, human health effects) soil (agricultural/homesteader and residential land use scenarios): ingestion and dermal exposure

Chemical	Target Hazard Quotient (THQ)	Target Cancer Risk (TCR)	Noncarcinogenic PRG(1)		Carcinogenic PRG(2)	
			Children	Adult	Children	Adult
INORGANICS (mg/kg)						
Arsenic	1.00	1.000E-06	5.88E+01	1.98E+02	8.72E-01	8.78E-01
Beryllium	1.00	1.000E-06	9.81E+02	3.29E+03	3.55E-01	3.57E-01
Cadmium	1.00	1.000E-06	1.96E+02	6.59E+02		
Chromium (III)	1.00	1.000E-06				
Copper	1.00	1.000E-06	7.26E+03	2.44E+04		
Lead	1.00	1.000E-06				
Manganese	1.00	1.000E-06	2.75E+04	9.22E+04		
Mercury	1.00	1.000E-06	5.88E+01	1.98E+02		
Nickel	1.00	1.000E-06	3.92E+03	1.32E+04		
Silver	1.00	1.000E-06	9.81E+02	3.29E+03		
Vanadium	1.00	1.000E-06	1.37E+03	4.61E+03		
Zinc	1.00	1.000E-06	5.88E+04	1.98E+05		
ORGANICS (mg/kg)						
Aroclor-1254	1.00	1.000E-06			1.48E-01	1.06E-01
Aroclor-1260	1.00	1.000E-06			1.48E-01	1.06E-01
Benzo(a)anthracene	1.00	1.000E-06	4.38E+03	1.05E+04	1.56E+00	1.12E+00
Benzo(a)pyrene	1.00	1.000E-06	4.38E+03	1.05E+04	1.56E-01	1.12E-01
Benzo(b)fluoranthene	1.00	1.000E-06	4.38E+03	1.05E+04	1.56E+00	1.12E+00
Benzo(k)fluoranthene	1.00	1.000E-06	4.38E+03	1.05E+04	1.56E+00	1.12E+00
Chrysene	1.00	1.000E-06	4.38E+03	1.05E+04	1.56E+01	1.12E+01
Dibenzo(a,h)anthracene	1.00	1.000E-06	4.38E+03	1.05E+04	1.05E-01	1.12E-01
Indeno(1,2,3-cd)pyrene	1.00	1.000E-06	4.38E+03	1.05E+04	1.56E+00	1.12E+00
RADIONUCLIDES (pCi/g)						
Americium 241		1.000E-06			9.95E+00	3.97E+00
Cesium 137+D		1.000E-06			8.52E+01	3.40E+01
Cobalt 60		1.000E-06			1.59E+02	6.35E+01
Uranium 234		1.000E-06			1.49E+02	5.95E+01
Uranium 235+D		1.000E-06			1.49E+02	5.95E+01
Uranium 238+D		1.000E-06			8.52E+01	3.40E+01

Table 7.2. Preliminary remediation goals (PRGs, human health effects) soil (open land use scenario): ingestion and dermal exposure

Chemical	Target Hazard Quotient (THQ)	Target Cancer Risk (TCR)	Noncarcinogenic PRG (1)		Carcinogenic PRG (2)	
			Children	Adult	Children	Adult
INORGANICS (mg/kg)						
Arsenic	1.00	1.000E-06	2.53E+02	4.61E+02	3.75E+00	2.05E+00
Beryllium	1.00	1.000E-06	4.22E+03	7.69E+03	1.53E+00	8.34E-01
Cadmium	1.00	1.000E-06	8.45E+02	1.54E+03		
Chromium (III)	1.00	1.000E-06				
Copper	1.00	1.000E-06	3.13E+04	5.69E+04		
Lead	1.00	1.000E-06				
Manganese	1.00	1.000E-06	1.18E+05	2.15E+05		
Mercury	1.00	1.000E-06	2.53E+02	4.61E+02		
Nickel	1.00	1.000E-06	1.69E+04	3.07E+04		
Silver	1.00	1.000E-06	4.22E+03	7.69E+03		
Vanadium	1.00	1.000E-06	5.91E+03	1.08E+04		
Zinc	1.00	1.000E-06	2.53E+05	4.61E+05		
ORGANICS (mg/kg)						
Aroclor-1254	1.00	1.000E-06			5.62E-01	2.48E-01
Aroclor-1260	1.00	1.000E-06			5.62E-01	2.48E-01
Benzo(a)anthracene	1.00	1.000E-06	1.67E+04	2.46E+04	5.92E+00	2.62E+00
Benzo(a)pyrene	1.00	1.000E-06	1.67E+04	2.46E+04	5.92E-01	2.62E-01
Benzo(b)fluoranthene	1.00	1.000E-06	1.67E+04	2.46E+04	5.92E+00	2.62E+00
Benzo(k)fluoranthene	1.00	1.000E-06	1.67E+04	2.46E+04	5.92E+00	2.62E+00
Chrysene	1.00	1.000E-06	1.67E+04	2.46E+04	5.92E+01	2.62E+01
Dibenzo(a,h)anthracene	1.00	1.000E-06	1.67E+04	2.46E+04	5.92E-01	2.62E-01
Indeno(1,2,3-cd)pyrene	1.00	1.000E-06	1.67E+04	2.46E+04	5.92E+00	2.62E+00
RADIONUCLIDES (pCi/g)						
Americium 241		1.000E-06			4.37E+01	9.26E+00
Cesium 137+D		1.000E-06			3.75E+02	7.94E+01
Cobalt 60		1.000E-06			7.00E+02	1.48E+02
Uranium 234		1.000E-06			6.56E+02	1.39E+02
Uranium 235+D		1.000E-06			6.56E+02	1.39E+02
Uranium 238+D		1.000E-06			3.75E+02	7.94E+01

Table 7.3. Preliminary remediation goals (PRGs, human health effects)
produce: ingestion exposure

Chemical	Target Hazard Quotient (THQ)	Target Cancer Risk (TCR)	Noncarcinogenic PRG		Carcinogenic PRG	
			Children	Adult	Children	Adult
INORGANICS (mg/kg soil)						
Arsenic	1.00	1.000E-06	1.11E+00	2.30E+00	1.64E-02	1.02E-02
Beryllium	1.00	1.000E-06	3.70E+02	7.67E+02	1.34E-01	8.32E-02
Cadmium	1.00	1.000E-06	5.91E-01	1.23E+00		
Chromium (III)	1.00	1.000E-06				
Copper	1.00	1.000E-06	2.19E+02	4.54E+02		
Lead	1.00	1.000E-06				
Manganese	1.00	1.000E-06	1.03E+02	2.15E+02		
Mercury	1.00	1.000E-06	2.96E-01	6.13E-01		
Nickel	1.00	1.000E-06	1.18E+02	2.45E+02		
Silver	1.00	1.000E-06	7.39E+00	1.53E+01		
Vanadium	1.00	1.000E-06	1.03E+03	2.15E+03		
Zinc	1.00	1.000E-06	2.22E+02	4.60E+02		
ORGANICS (mg/kg soil)						
Aroclor-1254	1.00	1.000E-06			1.72E-01	1.07E-01
Aroclor-1260	1.00	1.000E-06			3.04E-01	1.89E-01
Benzo(a)anthracene	1.00	1.000E-06	2.39E+03	4.95E+03	8.48E-01	5.28E-01
Benzo(a)pyrene	1.00	1.000E-06	4.71E+03	9.77E+03	1.67E-01	1.04E-01
Benzo(b)fluoranthene	1.00	1.000E-06	5.98E+03	1.24E+04	2.12E+00	1.32E+00
Benzo(k)fluoranthene	1.00	1.000E-06	8.34E+03	1.73E+04	2.96E+00	1.84E+00
Chrysene	1.00	1.000E-06	2.04E+03	4.22E+03	7.23E+00	4.50E+00
Dibenzo(a,h)anthracene	1.00	1.000E-06	5.24E+03	1.09E+04	1.86E-01	1.16E-01
Indeno(1,2,3-cd)pyrene	1.00	1.000E-06	2.45E+04	5.09E+04	8.71E+00	5.42E+00
RADIONUCLIDES (pCi/g soil)						
Cesium 137+D		1.000E-06			4.12E+00	9.53E-01
Cobalt 60		1.000E-06			7.69E+00	1.78E+00
Uranium 234		1.000E-06			1.08E+02	2.50E+01
Uranium 235+D		1.000E-06			1.08E+02	2.50E+01
Uranium 238+D		1.000E-06			6.18E+01	1.43E+01

Table 7.4. Preliminary remediation goals (PRGs, human health effects)
food chain: ingestion exposure

Chemical	Target Hazard Quotient (THQ)	Target Cancer Risk (TCR)	Noncarcinogenic PRG		Carcinogenic PRG	
			Children	Adult	Children	Adult
INORGANICS (mg/kg soil)						
Arsenic	1.00	1.000E-06	9.37E-01	1.95E+00	1.39E-02	8.64E-03
Beryllium	1.00	1.000E-06	2.36E+02	4.81E+02	8.52E-02	5.23E-02
Cadmium	1.00	1.000E-06	4.84E-01	1.10E+00		
Chromium (III)	1.00	1.000E-06	1.06E+04	2.53E+04		
Copper	1.00	1.000E-06	6.96E+01	1.98E+02		
Lead	1.00	1.000E-06				
Manganese	1.00	1.000E-06	9.11E+01	1.99E+02		
Mercury	1.00	1.000E-06	2.53E-01	5.43E-01		
Nickel	1.00	1.000E-06	9.27E+01	2.13E+02		
Silver	1.00	1.000E-06	1.96E+00	6.33E+00		
Vanadium	1.00	1.000E-06	2.04E+02	5.02E+02		
Zinc	1.00	1.000E-06		1.99E+02		
ORGANICS (mg/kg soil)						
Aroclor-1254	1.00	1.000E-06			3.79E-03	3.66E-03
Aroclor-1260	1.00	1.000E-06			1.52E-03	1.47E-03
Benzo(a)anthracene	1.00	1.000E-06	3.23E+02	9.89E+02	1.15E-01	1.05E-01
Benzo(a)pyrene	1.00	1.000E-06	1.27E+02	4.09E+02	4.52E-03	4.36E-03
Benzo(b)fluoranthene	1.00	1.000E-06	8.77E+01	2.83E+02	3.11E-02	3.02E-02
Benzo(k)fluoranthene	1.00	1.000E-06	5.13E+01	1.66E+02	1.82E-02	1.77E-02
Chrysene	1.00	1.000E-06	3.84E+02	1.15E+03	1.36E+00	1.22E+00
Dibenzo(a,h)anthracene	1.00	1.000E-06	1.08E+02	3.48E+02	3.84E-03	3.71E-03
Indeno(1,2,3-cd)pyrene	1.00	1.000E-06	8.46E+00	2.72E+01	3.00E-03	2.90E-03
RADIONUCLIDES (pCi/g soil)						
Americium 241		1.000E-06				
Cesium 137+D		1.000E-06			1.22E+00	3.90E-01
Cobalt 60		1.000E-06			2.12E+00	6.09E-01
Uranium 234		1.000E-06			3.54E+01	9.16E+00
Uranium 235+D		1.000E-06			3.54E+01	9.16E+00
Uranium 238+D		1.000E-06			2.02E+01	5.23E+00

Table 7.5. Preliminary remediation goals (PRGs, human health effects)
sediment: dermal exposure

Chemical	Target Hazard Quotient (THQ)	Target Cancer Risk (TCR)	Noncarcinogenic PRG		Carcinogenic PRG	
			Children	Adult	Children	Adult
INORGANICS (mg/kg)						
Arsenic	1.00	1.000E-06	3.73E+04	4.87E+04	5.52E+02	2.17E+02
Beryllium	1.00	1.000E-06	6.21E+05	8.12E+05	2.25E+02	8.81E+01
Cadmium	1.00	1.000E-06	1.24E+05	1.62E+05		
Chromium (III)	1.00	1.000E-06				
Copper	1.00	1.000E-06	>1E06	>1E06		
Lead	1.00	1.000E-06				
Manganese	1.00	1.000E-06	>1E06	>1E06		
Mercury	1.00	1.000E-06	3.73E+04	4.87E+04		
Nickel	1.00	1.000E-06	>1E06	>1E06		
Silver	1.00	1.000E-06	6.21E+05	8.12E+05		
Vanadium	1.00	1.000E-06	8.69E+05	>1E06		
Zinc	1.00	1.000E-06	>1E06	>1E06		
ORGANICS (mg/kg)						
Aroclor-1248	1.00	1.000E-06			1.25E+01	4.92E+00
Aroclor-1260	1.00	1.000E-06			1.25E+01	4.92E+00
Benzo(a)anthracene	1.00	1.000E-06	3.73E+05	4.87E+05	1.32E+02	5.19E+01
Benzo(a)pyrene	1.00	1.000E-06	3.73E+05	4.87E+05	1.32E+01	5.19E+00
Benzo(b)fluoranthene	1.00	1.000E-06	3.73E+05	4.87E+05	1.32E+02	5.19E+01
Benzo(k)fluoranthene	1.00	1.000E-06	3.73E+05	4.87E+05	1.32E+02	5.19E+01
Chrysene	1.00	1.000E-06	3.73E+05	4.87E+05	1.32E+03	5.19E+02
Dibenzo(a,h)anthracene	1.00	1.000E-06	3.73E+05	4.87E+05	1.32E+01	5.19E+00
Indeno(1,2,3-cd)pyrene	1.00	1.000E-06	3.73E+05	4.87E+05	1.32E+02	5.19E+01

Table 7.6. Preliminary remediation goals (PRGs, human health effects) surface water: incidental ingestion and dermal (swimming and wading) exposure

Chemical	Target Hazard Quotient (THQ)	Target Cancer Risk (TCR)	Noncarcinogenic PRG		Carcinogenic PRG	
			Children	Adult	Children	Adult
INORGANICS (μg/L)						
Aluminum	1.00	1.000E-06				
Barium	1.00	1.000E-06	3.00E+05	7.57E+05		
Chromium (III)	1.00	1.000E-06	>1E06	>1E06		
Cobalt	1.00	1.000E-06				
Copper	1.00	1.000E-06	1.58E+05	4.00E+05		
Manganese (water)	1.00	1.000E-06	2.14E+04	5.40E+04		
Mercury	1.00	1.000E-06	1.28E+03	3.24E+03		
Zinc	1.00	1.000E-06	>1E06	>1E06		
ORGANICS (μg/L)						
4,4-DDD	1.00	1.000E-06			1.26E+00	4.98E-01
4,4-DDT	1.00	1.000E-06	1.27E+01	1.67E+01	5.82E-01	2.29E-01
Acetone	1.00	1.000E-06	3.23E+05	6.63E+05		
Aldrin	1.00	1.000E-06	8.23E+01	1.56E+02	1.26E+00	7.13E-01
Bis(2-ethylhexyl)phthalate	1.00	1.000E-06	6.22E+03	8.43E+03	1.73E+02	7.02E+01
Chloroform	1.00	1.000E-06	9.73E+03	1.43E+04	1.24E+03	5.47E+02
Dieldrin	1.00	1.000E-06	2.99E+01	4.19E+01	2.91E-01	1.22E-01
Diethyl phthalate	1.00	1.000E-06	>1E06	>1E06		
Endosulfan	1.00	1.000E-06				
Endosulfan Sulfate	1.00	1.000E-06				
Endrin	1.00	1.000E-06	1.79E+02	2.51E+02		
Fluoranthene	1.00	1.000E-06	1.21E+03	1.59E+03		
gamma-BHC	1.00	1.000E-06	2.01E+02	2.85E+02	4.01E+00	1.70E+00
Heptachlor Epoxide	1.00	1.000E-06	4.21E+00	5.71E+00	2.77E-01	1.13E-01
Tetrachloroethylene	1.00	1.000E-06	2.18E+03	2.93E+03		
1,1,1-Trichloroethane	1.00	1.000E-06	5.10E+04	7.13E+04		
Trichloroethylene	1.00	1.000E-06				
RADIONUCLIDES (pCi/L)						
Americium 241		1.000E-06			2.23E+02	6.68E+01
Cesium 137+D		1.000E-06			1.91E+03	5.72E+02
Cobalt 60		1.000E-06			3.56E+03	1.07E+03
Neptunium 237+D		1.000E-06			2.43E+02	7.28E+01
Uranium 234		1.000E-06			3.34E+03	1.00E+03
Uranium 235+D		1.000E-06			3.34E+03	1.00E+03
Uranium 238+D		1.000E-06			1.91E+03	5.72E+02

Table 7.7. Preliminary remediation goals (PRGs, human health effects) groundwater: ingestion and inhalation exposure[a]

Chemical	Target Hazard Quotient (THQ)	Target Cancer Risk (TCR)	Noncarcinogenic PRG		Carcinogenic PRG		MCL[b]	PMCL[b]	MCLG[b]	PMCLG[b]
			Children	Adult	Children	Adult				
INORGANICS (μg/L)										
Arsenic	1.00	1.000E-06	9.24E+00	1.10E+01	1.37E-01	4.87E-02	50			
Beryllium	1.00	1.000E-06	1.54E+02	1.83E+02	5.57E-02	1.98E-02		1		0
Cadmium (water)	1.00	1.000E-06	1.54E+01	1.83E+01			10			
Chromium (III)	1.00	1.000E-06					50			
Copper	1.00	1.000E-06	1.14E+03	1.35E+03			1000	1300		1300
Lead	1.00	1.000E-06					50	5		
Manganese (water)	1.00	1.000E-06	1.54E+02	1.83E+02			50			
Mercury	1.00	1.000E-06	9.24E+00	1.10E+01			2			
Nickel	1.00	1.000E-06	6.16E+02	7.30E+02				1		1
Vanadium	1.00	1.000E-06	2.16E+02	2.56E+02						
Zinc	1.00	1.000E-06	9.24E+03	1.10E+04			5000			
ORGANICS (μg/L)										
Acetone	1.00	1.000E-06	3.08E+03	3.65E+03						
Methylene chloride	1.00	1.000E-06	1.83E+03	2.18E+03	3.10E+01	1.13E+01		5		0
RADIONUCLIDES (pCi/L)										
Radium 228+D		1.000E-06			3.61E+00	4.76E-01		2-20		0
Uranium 234		1.000E-06			2.25E+01	2.98E+00		5-40		0
Uranium 235+D		1.000E-06			2.25E+01	2.98E+00		5-40		0
Uranium 238+D		1.000E-06			1.29E+01	1.70E+00		5-40		0

[a]PRG based on ingestion and inhalation where reference concentrations (RfCs) and cancer slope factors for inhalation route were available.

[b]Maximum contaminant levels (MCLs), maximum contaminant level goals (MCLGs), proposed maximum contaminant levels (PMCLs), and proposed maximum contaminant level goals (PMCLGs).

Applicable or relevant and appropriate requirements (ARARs) and to-be-considered (TBC) guidance May 1992, Prepared by: ORNL, Under DOE Contract No. DE-AC05-84OR21400.

Table 7.8. Preliminary remediation goals[a] for principal contaminants of concern identified following the baseline human health risk assessment

Chemical	Soil ingestion/dermal contact				Produce ingestion		Dermal contact with sediment		Ingestion and dermal contact with surface water		Groundwater ingestion	
	Residential/agricultural land use areas		Open land use areas		Residential/agricultural land use areas		Residential/agricultural land use areas		Residential/agricultural land use areas		Residential/agricultural land use areas	
	Noncarc. effects	Carc. effects	Noncarc. effects	Carc. effects	Noncarc. effects	Carc. effects	Noncarc. effects	Carc. effects	Noncarc. effects	Carc. effects	Noncarc. effects	Carc. effects
Children												
Arsenic	5.88E+01	8.72E-01	2.53E+02	3.75E+00	1.11E+00	1.64E-02	3.73E+04	5.52E+02	NA[b]	NA[b]	9.24E+00	1.37E-01
Cadmium	1.96E+02		8.45E+02		5.91E-01		1.24E+05		NA[b]		NA[b]	
Mercury	5.88E+01		2.53E+02		2.96E-01		3.73E+04		1.28E+03		2[c]	
Adults												
Arsenic	1.98E+02	8.78E-01	4.61E+02	2.05E+00	2.30E+00	1.02E-02	4.87E+04	2.17E+02	NA[b]	NA[b]	1.10E+01	4.87E-02
Cadmium	6.59E+02		1.54E+03		1.23E+00		1.62E+05		NA[b]		NA[b]	
Mercury	1.98E+02		4.61E+02		6.13E-01		4.87E+04		3.24E+03		2[c]	

[a]preliminary remediation goals were derived using target hazard quotients (THQs) of 1.00 and target cancer risks (TCRs) of 1.00E-06 for each contaminant
[b]not a COC element in respective media
[c]the Safe Drinking Water Act maximum contaminant goal will be used as a preliminary remediation goal for mercury

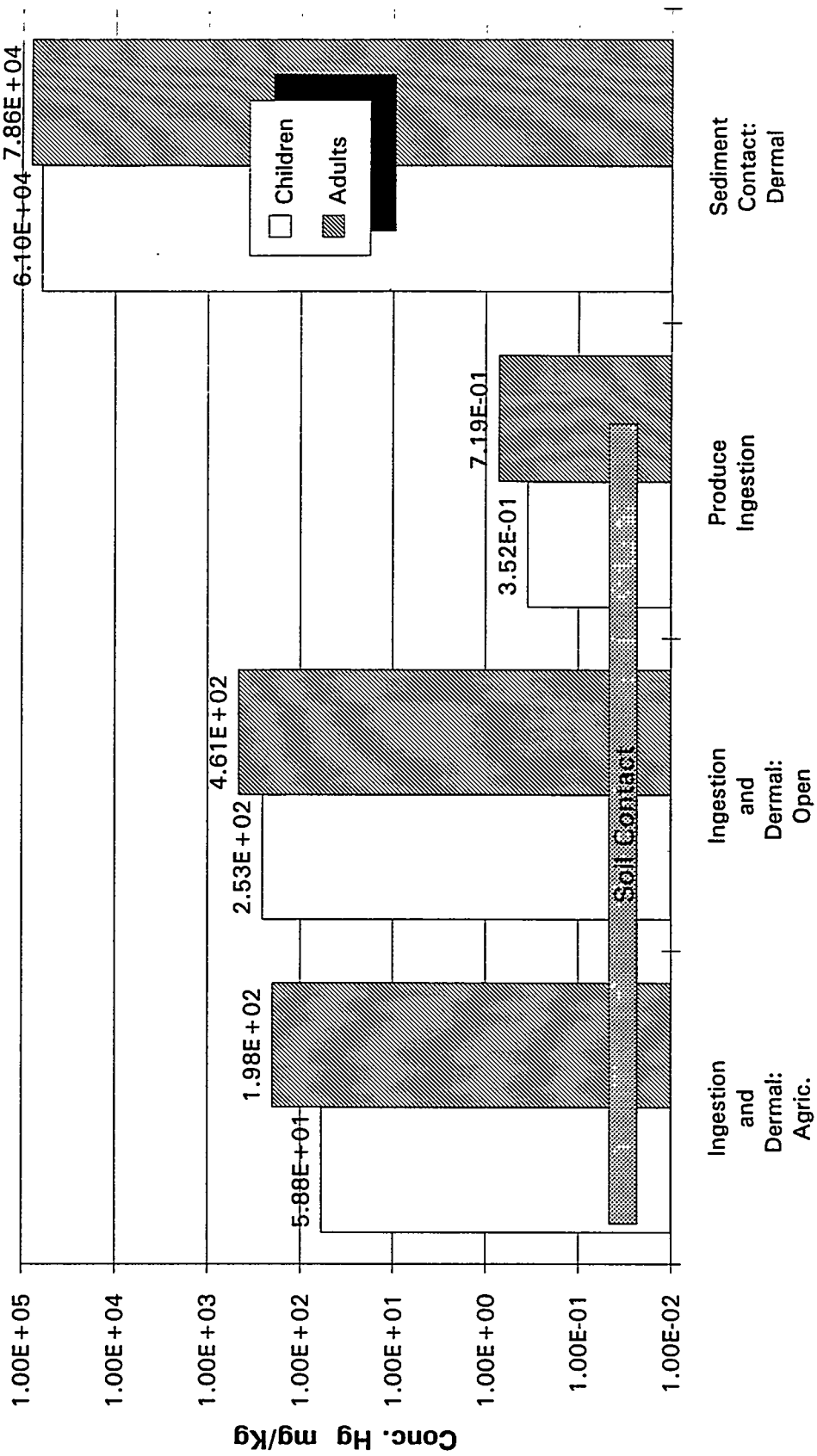

Fig. 7.1. Human health PRGs for mercury in soils and sediment of EFPC.

7.3 DERIVATION OF ECOLOGICALLY BASED PRGS FOR EFPC

7.3.1 Introduction

According to EPA guidance, PRGs must, at a minimum, comply with chemical-specific ARARs and provide overall protection of the environment. To do so, two kinds of PRGs are derived: (1) concentrations based on ARARs and (2) risk-based concentrations. The former will fulfill the regulatory requirement that remedial activities meet ARARs, and the latter will address the requirement that remedial activities must be protective of the environment. Since the assessment endpoints of the baseline ecological risk assessment are also based on ARARs and ecological risk, all assessment endpoints should be met as a result of reducing contaminant concentrations to the levels of PRGs.

Chemical-specific ARARs (Sect. 4.1.1) have been established to define the maximum allowable levels of contamination of physical media that are consistent with the protection of animal and plant populations and with maintenance of the ecosystem. Chemical-specific ARARs are presented in Table 4.1. ARARs may apply to air, surface water, groundwater, sediment, or soil; each of these media will be discussed below. In addition, To Be Considered (TBC) guidance will be discussed where appropriate.

The PRGs presented here are based on (1) analytical data obtained during sampling of biota, (2) physical media sampling carried out during Phase Ia and Phase Ib, (3) published information about the toxicity of contaminants of concern, and (4) relationships among these findings. It is assumed that the contaminant concentrations measured during sampling of aquatic biota are representative of the associated sampling locations, although it is recognized that contaminant concentrations in water, as well as population distributions and community structure, are variable over time. As is the case with PRGs for human health, the ecological PRGs are based on single chemicals and do not yet take into consideration combined exposure and potential antagonistic or synergistic effects among contaminants.

Potential ecological exposure routes for contaminants in EFPC and the EFPC floodplain have been described in the EFPC RI Report. They are shown schematically in Figs. 6.38 (Y-12 Plant contaminants) and 6.39 (EFPC floodplain contaminants). These figures show that some transport pathways and some exposure routes are more important than others in ecological exposure to contaminants. Exposures through contaminated physical media may be reduced by cleanup of the media; PRGs are to be established that will ensure that residual contaminant levels will be

protective of ecological resources. Exposure may occur by ingestion of or dermal contact with soil, inhalation of vapors from soil, ingestion of or direct uptake from water from contaminated seeps or surface water from EFPC, or ingestion of contaminated food.

EPA has provided guidance for calculating PRGs for human health by a variety of exposure pathways (EPA 1991a), but analogous guidance for non-human biota is not available. Thus, a major limitation to the derivation of ecological PRGs is that exposure routes are partially defined. For example, a quantitative linkage between whole-body concentration of a contaminant in a small mammal and its concentration in the soil near where the animal was trapped has yet to be established. Also, data on exposure rates and toxicity for terrestrial animals are limited. Therefore, there are several limitations associated with the derivation of ecological PRGs.

Historic evidence and evaluation of the nature and extent of contamination (Sect. 5.1) showed that mercury and cadmium are significant potential toxicants to ecological receptors in EFPC and the EFPC floodplain. PCBs and PAHs were also shown to be present in EFPC sediments and floodplain soils. Pesticides are known from historical data to have been used at the Y-12 Plant and are likely candidates for surface runoff from other locations in the EFPC watershed. PRGs for mercury and cadmium are evaluated in the following subsections, as are compounds or mixtures chosen to represent PCBs, PAHs, and pesticides. These five chemicals or chemical classes are judged to be the most important contributors to ecological risk from chemicals. Additional metals will be discussed as well.

Because exposures of biota in the EFPC floodplain to these contaminants may potentially be derived from five major sources—air, surface water, groundwater, instream sediments, and soils—these media will be considered separately in the discussion of PRGs.

7.3.2 Air

Air is not considered a major pathway of ecological exposure to contaminants in EFPC or the EFPC floodplain. Concentrations of mercury in ambient air at the EFPC floodplain have been shown to be low (Sects. 3.2.8, 6.2.1.1, Appendix H). In addition, airborne contaminants in the EFPC floodplain are transient, having other environmental media as their sources. Cleanup of the source media rather than of air would be required to reduce airborne exposures to contaminants in the EFPC floodplain. Therefore, remedial goals for air are not being proposed.

7.3.3 Surface Water

Surface water has been shown to be a major pathway transporting dissolved and particulate contaminants to aquatic biota in EFPC, either directly or through the food chain. Numerous investigators have concluded that ongoing releases from the Y-12 Plant are the major source of water-borne contaminants (Van Winkle et al. 1984, Loar 1992, Hinzman 1992, Sect. 6.4.1.2). These releases appear to include mercury and other metals, PCBs, PAHs, pesticides, and chlorine residuals (Sect. 6.4.1.2).

Even though water-borne releases from the Y-12 Plant may be the major source of contaminants in EFPC surface water, it is likely that there are also risks due to transport of contaminants from soil and sediment to surface water. To provide background for discussions of PRGs related to transport of particulate-bound contaminants from sediment and soil to surface water, potential PRGs for surface water are discussed in the following subsections.

Attainment of some PRGs for surface water can be assessed more reliably by measuring contaminant concentrations in biota than by analyzing the water itself. Concentrations of contaminants in EFPC surface water are sufficiently high to cause decreases in species richness and species diversity, changes in fish community structure and population distributions, and increases in contaminant body burdens. However, it is important to understand that for some analytes, the concentrations that cause such biological effects are lower than the quantitation limits. Many analytes accumulated in aquatic biota in EFPC, even though their observed concentrations in surface water were below detection limits. Therefore, accumulation in biota is a more reliable indication of their presence than is chemical analysis of the abiotic media. Similarly, if PRGs are chosen to be equal to ARARs for surface water, attainment of some of those PRGs may not be measurable, because ARARs for many contaminants in surface water are below detection limits of standard analytical techniques. For example, the ambient water quality criterion (AWQC) for protection of freshwater fish from chronic exposure to mercury is 0.012 μg/L, and AWQCs for pesticides range from 0.001 to 0.004 μg/L (Table 4.1). The contract-required detection limit for mercury in water is 0.2 μg/L, and detection limits for pesticides are 0.05 to 0.10 μg/L.

Regardless of detection limits, PRGs for surface water should be chosen so that ARARS are met and so that deleterious effects are mitigated. Although mercury, PCBs, pesticides, and PAHs have been implicated as potential ecological COCs, the specific stressors that cause changes in community structure and decreases in taxonomic richness and diversity (Sects. 6.3.3.1 and

6.3.3.2) have been only partially identified. Therefore, continued surveillance of aquatic populations will be necessary to determine whether attainment of remedial goals for specific chemicals has the required effect of mitigating deleterious impacts on aquatic populations. The discussion of surface water PRGs will focus on ARARs and TBC guidance, protection of EFPC biota from contaminant toxicity, and protection of predators from contaminant toxicity.

7.3.3.1 AWQCs as PRGs

The most stringent ARAR for surface water is attainment of AWQCs for protection of freshwater biota. This criterion is intended to protect freshwater organisms from lifetime accumulation of contaminants from water and considers bioconcentration of compounds from the water column. The following subsections evaluate AWQCs as potential PRGs for surface water.

Mercury. The AWQC for mercury [0.012 μg/L (Table 4.1)] was exceeded by a factor of 45 in the sample taken at Site 1 (Table 6.49). The concentration of water-borne mercury was 0.54 μg/L at Site 1 and decreased with distance downstream, eventually becoming undetectable (<0.2 μg/L) (Table 6.49). Because (1) the Y-12 Plant is a known mercury source, (2) there are no other known sources upstream of Site 1, and (3) instream sediments have been shown not to serve as a major current mercury source to surface water (Sect. 6.3.2.3), it was concluded that ongoing releases from the Y-12 Plant are the major source of mercury in aquatic biota. Therefore, setting the PRG to the AWQC for mercury would require a reduction in mercury release from the Y-12 Plant.

Cadmium and Other Inorganic Contaminants. The AWQC for cadmium (1.0 μg/L) was below the analytic detection limit (5 μg/L). Therefore, the concentration of water-borne cadmium could exceed the AWQC by as much as a factor of 5. However, cadmium was undetectable in 30 of 32 EFPC samples of aquatic biota, indicating that concentrations available for uptake by biota are sufficiently low to preclude bioconcentration to detectable levels in EFPC biota.

AWQCs for arsenic, chromium, copper, nickel, and zinc were not exceeded (Table 6.49). The AWQC for lead (3.0 μg/L) was exceeded by a factor of 1.03 at Sites 1 and 3. Further sampling would be required to validate these findings and justify remedial actions to reduce lead levels to the AWQC.

PCBs. The AWQC for chronic exposure of freshwater biota to PCBs is 0.001 μg/L (Table 4.1). PCBs were not detected in surface water samples taken during biota sampling

(Sect. 6.2.3.2). However, the contract-required detection limit for PCBs in water is 0.5 μg/L; at the contract-required detection limit, undetected concentrations of PCBs in surface water could exceed the AWQC by a factor of 500. The accumulation of PCBs in fish and crayfish shown in Sect. 6.2.3.2 indicates that PCBs are present in EFPC and must be monitored by further surveillance of aquatic biota.

Bioconcentration factors (BCFs) can be used to estimate the concentration of contaminants in fish flesh corresponding to the AWQC. BCFs for PCBs such as Aroclor 1254 and Aroclor 1260 have been reported to be in the range of 49,000 L/kg to approximately 100,000 L/kg (Lyman et al. 1990). Using these values, the concentration of PCBs in fish flesh corresponding to a concentration of 0.001 μg/L can be calculated:

$$C_{biota} = C_{water} \times BCF = 0.001\ \mu g/L \times 49{,}000 \text{ to } 100{,}000\ L/kg$$
$$= 49 \text{ to } 100\ \mu g/kg = 0.05 \text{ to } 0.1\ mg/kg$$

These values might be used as indicators for attainment of the AWQC for PCBs. Maximum Aroclor 1260 concentrations of 8.1 mg/kg in stonerollers and 2.8 mg/kg in redbreast sunfish were observed in EFPC samples, indicating that the surface water concentration was probably significantly above the AWQC.

Chlordane. For purposes of deriving PRGs for pesticides, we chose chlordane as representative of the pesticides because it caused the highest pesticide body burden in stonerollers and redbreast sunfish. Chlordane has been shown to adversely affect sensitive species of fish and aquatic invertebrates at nominal water concentrations between 0.2 and 3.0 μg/L (Eisler 1990).

Pesticides were not detected in surface water samples taken during the biota sampling (Sect. 6.2.3.2) at the contract-required detection limit of 0.5 μg/L. Chlordane, dieldrin, and heptachlor were detected in whole-body samples of redbreast sunfish (maximum of ~60 μg/kg) and stonerollers (maximum of approximately 100 μg/kg for chlordane and dieldrin, approximately 45 μg/kg for heptachlor). In crayfish chlordane, dieldrin, and DDE were detected at a maximum of approximately 10 μg/kg.

No AWQC for chlordane has been set. Although not an ARAR, the recommended freshwater aquatic life protection criterion is 0.0043 μg/L (24 hour average); chlordane in fresh water is not to exceed 2.4 μg/L at any time (Eisler 1990). The concentration of chlordane in fish

at the chronic criterion concentration can be calculated from a BCF of 37,800 (Lyman et al. 1990):

$$C_{biota} = C_{water} \times BCF = 0.0043\ \mu g/L \times 37{,}800\ L/kg$$
$$= 160\ \mu g/kg$$

This value might be used as an indicator for attainment of the recommended surface water chronic concentration limit for chlordane. Body burdens of chlordane in fish seem to indicate that concentrations of chlordane are below that limit in EFPC surface water.

PAHs. AWQCs for PAHs have not been established. The most stringent ARAR for PAHs in water is a proposed MCL of 0.03 $\mu g/L$ for chrysene and pyrene (Table 4.1). PAHs were not detected in surface water samples taken during biota sampling (Sect. 6.2.3.2). At the contract-required detection limit of 10 $\mu g/L$ in water, concentrations of these undetected analytes could exceed the MCL by a factor of 300. No criteria for protection of fish and aquatic life are listed as ARARs for PAHs.

BCFs for organic chemicals have been shown to be related to their octanol-water partition coefficients (K_{ow}) by the relationship:

$$\log BCF = 0.76 \log K_{ow} - 0.23$$

[derived from data for 84 compounds, $r^2 = 0.82$ (Veith et al. 1980)]. BCFs for PAHs ranging from approximately 300 to approximately 115,000 have been calculated from their octanol-water partition coefficients (K_{ow}). For example, using the observed maximum of 18 μg benzo(a)anthracene/kg in stonerollers and the reported BCF for benzo(a)anthracene of 11,700, the benzo(a)anthracene concentration in the water column corresponding to the observed body burdens was calculated:

$$C_{biota} = C_{water} \times BCF$$
$$= 0.1\ \mu g/L \times 11{,}700\ L/kg$$
$$= 1170\ \mu g/kg$$

Similar calculations for other selected PAHs are presented in Table 6.73. None of the PAHs whose concentrations were reported in aquatic biota appears to exceed ARARs in EFPC surface water.

7.3.3.2 FDA action limits for fish and shellfish

FDA action limits for contaminants in fish and shellfish (49 *FR* 21514; 55 *FR* 14359) are not intended to be protective of aquatic biota, nor are they strictly ARARs for environmental cleanup. However, it is a goal of the remedial program to restore the recreational fishery. Because the action limits are defined for aquatic biota, they will be addressed in this subsection of the discussion of PRGs.

FDA action limits will be considered for redbreast sunfish and crayfish rather than for stonerollers, since stonerollers are not considered edible fish. A comparison of mercury concentrations in whole-body homogenates and fillets of redbreast sunfish (Table 7.9) shows that whole-body concentrations of mercury were similar to concentrations in fillets, whereas whole-body concentrations of PCBs were higher than concentrations in fillets by an average factor of approximately 5 (Table 7.9). Because small pan fish are not necessarily filleted before they are cooked, this evaluation is based on whole-body contaminant concentrations.

Mercury. Although the route of exposure to mercury has not clearly been established, it is assumed for these calculations that water-borne mercury is the ultimate source of exposure, either by direct uptake or through food [redbreast sunfish at Site 3 may be an exception (see Sect. 6.2.3.2)]. Because of the edible aquatic biota sampled, crayfish had the highest mercury body burdens and showed the most direct correlation of body burden with surface water mercury concentrations, they were used to calculate a range of surface water PRGs for mercury as shown below.

Two approaches were taken to calculate surface water PRGs: in the proportional method, it is assumed that the mercury body burden is proportional to surface water concentration, i.e., both uptake and elimination rates are proportional to surface water concentration. In the threshold method, it is assumed that there is a limit to the rate at which mercury can be eliminated from the body, whereas the rate of uptake is proportional to the surface water concentration. Therefore, in the latter case there is a threshold concentration below which the body burden reflects the balance between uptake and depuration, and above which depuration is not able to balance uptake, so the body burden rises more rapidly with increasing surface water concentration. This threshold effect is shown in Fig. 7.2, which shows that body burden rose slowly with increasing concentrations at surface water concentrations below approximately 0.3 μg/L and much more rapidly at higher concentrations.

Table 7.9. Relative contaminant concentrations in whole-body homogenates and fillets of redbreast sunfish in EFPC

Site	Tissue Type	Analytes[a]				
		Mercury	Cadmium	Chlordane[b]	PCB	PAH
3	Fillet	1.765	BDL[c]	0.009	0.71	BDL
	Whole-body	2.184	BDL	0.055	2.04	BDL
	Ratio	**0.81**	--	**0.16**	**0.35**	--
5	Fillet	0.722	BDL	0.013	0.17	BDL
	Whole-body	0.476	BDL	0.083	0.99	BDL
	Ratio	**1.51**	--	**0.16**	**0.17**	--

[a] Average concentrations (mg/kg)
[b] Alpha chlordane plus gamma chlordane
[c] Below detection limits

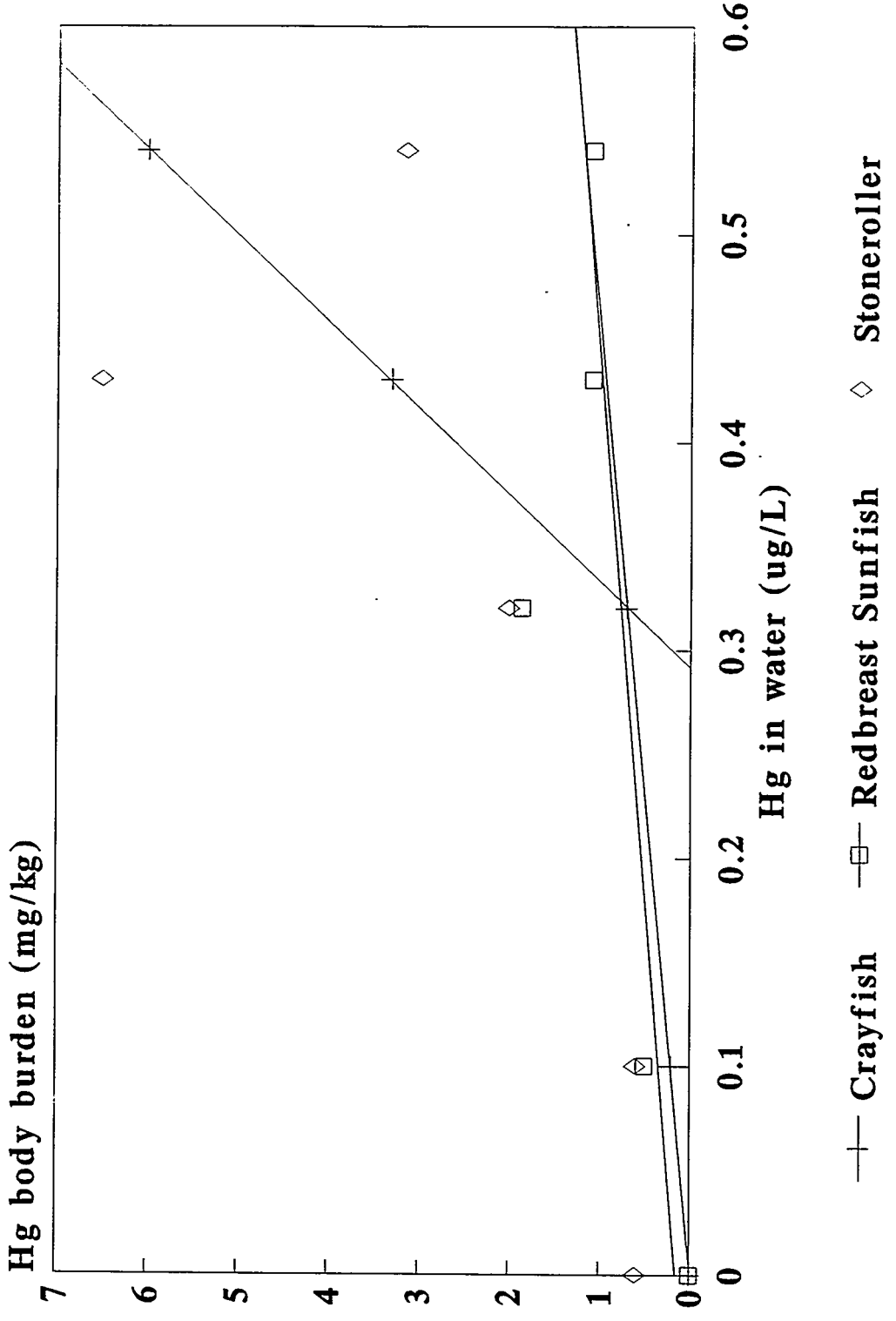

Fig. 7.2. Correlation of mercury body burden in redbreast sunfish, stonerollers, and crayfish with total mercury in EFPC surface water.

At Site 1, the observed concentration of water-borne mercury was 0.54 μg/L and the crayfish mercury body burden exceeded the FDA action limit by 6-fold. By the proportional method, if water-borne mercury is the principal source to crayfish, the FDA action limit should be met by a 6-fold reduction in concentration, to 0.09 μg/L. In contrast, Fig. 7.2 shows that a surface water concentration of 0.34 μg/L corresponds to 1 mg/kg mercury in the whole crayfish. The maximum mercury body burden in redbreast sunfish (1.8 mg/kg) occurred at Site 3, where the surface water concentration was 0.32 μg/L. By the proportional method, reducing the concentration to 0.18 μg/L would be expected to reduce the redbreast sunfish body burden to 1 mg/kg. However, mercury body burden in redbreast sunfish at Site 3 may have other sources in addition to surface water. The remainder of the mercury body burden data for redbreast sunfish suggest that the FDA action limit would be met by concentrations below approximately 0.4 μg/L (Table 7.2). Therefore, a range of 0.09 to 0.4 μg mercury/L is proposed as a conservative PRG range for reduction of body burdens in edible aquatic biota to achieve FDA action limits for mercury. Reduction in surface water mercury concentration to 0.012 μg/L to meet AWQCs should reduce mercury concentrations in fish and shellfish well below the FDA action limits.

Cadmium. FDA has not established an action limit for cadmium in food for human consumption.

PCBs. The concentration of PCBs in water that would be expected to lead to a body burden of 2 mg/kg in biota can be calculated by using the BCF. BCFs for PCBs such as Aroclor 1254 and Aroclor 1260 have been reported to be in the range of 49,000 to approximately 100,000 (Lyman et al. 1990). Using the more conservative of these values, the calculated maximum allowable concentration of PCBs in surface water would be:

$$2000\ \mu g/kg\ /\ 100{,}000\ L/kg = 0.02\ \mu g/L$$

This result is above the AWQC for PCB by a factor of 20.

A reduction of approximately 75% in water-borne PCB concentrations should allow attainment of the FDA action limit for PCB in edible fish and shellfish, and attainment of the AWQC for PCBs [0.001 μg/L (Table 4.1)] is expected to reduce whole-body concentrations of PCB below FDA action limits. However, since the current levels of water-borne PCB in EFPC are below detection limits (Sect. 6.2.3.2), documenting attainment of this PRG would require continued surveillance of PCBs in fish rather than in water.

Chlordane and Other Pesticides. Action limits for pesticides in edible portions of fish are presented in 55 *FR* 14359 (corrected 55 *FR* 30796). The FDA action limit for chlordane in food for human consumption is 0.3 mg/kg fresh weight (55 *FR* 14359). The concentration of chlordane in surface water associated with bioconcentration to the FDA action limit was calculated using a published BCF value of 37,800 for chlordane 37,800 (Lyman et al. 1990). The result was:

300 μg/kg / 37,800 L/kg = 0.008 μg/L

This result is above the recommended limit (Eisler 1990) of 0.0043 μg/L for chlordane in surface water, which would therefore be protective.

PAHs. FDA has not set action limits for PAHs in food for human consumption.

7.3.3.3 Protection of biota from contaminant toxicity

An exposed organism may accumulate chemicals above threshold concentrations that cause damage to essential body functions, to reproduction, or to behaviors essential for survival. These concentrations are both chemical-specific and organism-specific. Biota may be very sensitive to toxic compounds during certain developmental periods, and it is necessary to account for sensitive life stages. To calculate the tolerable concentration of a contaminant in environmental media, it is necessary to know either what environmental concentration is toxic or how much the contaminant is concentrated and what body burdens are associated with toxicity. PRGs for protection of biota from toxicity of contaminants in surface water are considered in the following subsections.

Mercury. Mercury is toxic to larval rainbow trout at concentrations below 0.1 μg/L, and sublethal effects on reproduction by aquatic biota have been observed at concentrations from 0.03 to 0.1 μg/L (Eisler 1987a). A limit of 5 mg mercury/kg body weight has been proposed as a conservative level to protect aquatic biota from these toxic effects (Eisler 1987a). Figure 7.2 shows that the surface water mercury concentration corresponding to 5 mg/kg in whole crayfish is 0.5 μg/L, whereas the ratio method yields a PRG of 0.54/1.2 = 0.45 μg/L. Reducing mercury concentrations in surface water to the AWQC concentration of 0.012 μg/L should reduce body burdens in the aquatic biota well below the level of expected toxicity.

Cadmium. The recommended upper limit for cadmium in drinking water for humans is 10 μg/L. This is not sufficient to protect many species of freshwater biota against biocidal properties of cadmium or against sublethal effects, such as reduced growth and inhibited reproduction (Eisler 1985). The ambient water quality criteria formulated for the protection of freshwater aquatic life state that for total recoverable cadmium, the criterion is a function of the water hardness. Based on the data in Table 6.44, the criteria range from 3.9 μg/L at Sites 2 and 5 to 4.6 μg/L at Site 4. The Hinds Creek reference site, because of its high alkanet, would require a protection criterion of 6.3 μg/L. According to the US Fish and Wildlife Service (FWS) (Eisler 1985), new data suggest that these criteria are not always protective of the most sensitive freshwater species. The report suggests that cadmium water concentrations in excess of 3 μg/L may be hazardous to aquatic biota and that levels near 1 μg/L should be cause for concern in waters with low alkanet. Because alkanet in EFPC surface water is relatively high (Table 6.44), the alkalinity-dependent AWQC for cadmium (1 μg/L) is accepted as the PRG for surface water (see Sect. 7.3.3.1).

PCBs. A concentration of 0.1 μg/L has been shown to adversely affect the growth of freshwater algae and fish (EPA 1990). Whole body residues of 0.4 mg PCB/kg fresh weight are associated with reproductive toxicity in rainbow trout and hence have been proposed by EPA as a conservative limit for the protection of aquatic species (Eisler 1986). The concentration of PCBs in surface water expected to result in a whole-body concentration of 400 μg/kg was calculated using the BCF of 100,000. This value was 0.004 μg/L, four times the AWQC for PCBs. Therefore, attaining the AWQC should be protective against toxicity to aquatic biota.

Chlordane. Nominal water concentrations of chlordane between 0.2 and 3.0 μg/L have been shown to cause adverse effects in aquatic organisms (Eisler 1990). These values are much higher than the recommended limit of 0.0043 μg/L, which would therefore be protective.

PAHs. Fluorene at 500 μg/L was reported to be lethal to 12% of exposed fish (Eisler 1987). An arbitrary safety factor of 100 would lead to a recommended maximum concentration of 5 μg/L. This value is more than 10-fold above ARARs for PAHs in surface water (Table 4.1), which would therefore be protective of aquatic biota.

7.3.3.4 Protection of predators from contaminant toxicity

Contaminants in prey species accumulate in predators if they are not excreted or metabolized as rapidly as they are ingested. Accumulation of metals may cause failure or dysfunction of

organs, leading to death, susceptibility to disease, altered behavior incompatible with survival, or compromised reproductive capacity.

PRGs for protection of predators are evaluated by considering body burdens of contaminants in prey species that are known to cause toxicity in predators, and then calculating what contaminant levels in abiotic media are expected to prevent contaminant accumulation in the prey species above those protective levels. In these calculations composition of the diet and the feeding range of the predators may be considered as well. For surface water, it is conservatively assumed that the feeding ranges of piscivorous predators are restricted to EFPC.

Mercury. On the basis of toxicological data, the criterion recommended by Eisler for protection of predators from mercury in prey was set at 0.05 mg/kg diet for piscivorous birds and 1.1 mg/kg diet for small mammals (Eisler 1987a). In general, a gradient of mercury concentrations in aquatic biota was observed, decreasing from the sampling location nearest the Y-12 Plant (Sect. 6.2.3.2).

Mercury body burdens in crayfish correlated well with mercury concentrations in surface water (Fig. 7.2; Sect. 6.2.3.2). If surface water is the primary source to crayfish, the ratio method shows that a reduction of crayfish body burden from 6 mg/kg to 0.05 mg/kg would require a proportional decrease of 120-fold in maximum mercury concentration in water, to 0.54/120 = 0.0045 μg/L. By the threshold method, the concentration in surface water corresponding to a body burden of 0.05 μg/L is 0.022 μg/L. The AWQC for mercury (0.012 μg/L) is between the calculated PRG values. Because the threshold method better represents observed contaminant uptake, it is likely that meeting the AWQC should suffice to reduce body burdens in aquatic biota below the target concentration.

Stonerollers accumulated high levels of mercury, presumably by ingestion of contaminated food, predominantly periphyton. Figure 7.2 shows that mercury body burdens in stonerollers were not correlated with surface water mercury concentration. Furthermore, BCFs for accumulation of mercury by periphyton were not available. Therefore, these data were not used to calculate a surface water PRG.

Cadmium. Among all of the samples of aquatic biota taken from EFPC, cadmium was detected in only one sample of crayfish. Therefore, cadmium is not being bioconcentrated significantly from EFPC surface water. This implies that the AWQC for cadmium (Sect. 7.3.3.1) is sufficiently protective of predators on aquatic biota for toxicity from cadmium exposure.

PCBs. O'Connor and Pizza (1984) recommended PCB levels in fish diets of less than 0.5 mg/kg fresh weight based on their investigations with striped bass (Eisler 1986). The concentration of PCBs in surface water leading to a body burden of 500 μg/kg in aquatic prey was calculated using a BCF for Aroclor 1260 of 100,000 (Lyman et al. 1990) and found to be 0.005 μg/L, five times the AWQC. Therefore, the AWQC is protective for piscivorous biota.

In the case of birds, a diet containing 3 mg PCB/kg fresh weight has been shown to result in high PCB concentration in the eggs of screech owls (McLane and Hughes 1984). If similar biotransfers occur for piscivorous birds, consumption of stonerollers, the predominant fish near Site 1, could result in excessive levels of PCBs in eggs and potential deleterious effects to offspring. The calculated surface water concentration yielding body burdens of 3 mg/kg is 3 mg/kg / 100,000 L/kg = 0.03 μg/L. Therefore, attainment of the AWQC for PCBs in surface water would be protective against aquatic toxicity.

The AWQC for PCBs should be protective for piscivorous birds, but it might not protect mink eating EFPC fish. The mink is very sensitive to PCBs, with death documented at 100 μg/kg fresh weight of diet (Aulerich et al. 1985). Using the BCF of 100,000 and a concentration of 0.001 μg/L (the AWQC), the calculated body burden for prey fish would be 100 μg/kg, 30-fold below the concentration shown to cause accumulation in owl eggs but not below the level of potential lethality to mink.

Chlordane. FWS (Eisler 1990) lists the guideline for the protection of predatory fish from chlordane at 0.1 mg/kg fresh weight. Sensitive bird species had reduced survival after consumption of diets with 1.5 mg chlordane/kg of ration, or after a single oral dose of 14.1 mg/kg body weight (BW). FWS did not derive criteria for protection of mammalian wildlife and birds. Additional NOEL data are needed to develop these guidelines.

Using 0.1 mg/kg fresh weight as a conservative value, and the BCF value for chlordane of 37,800 (Lyman et al. 1990), the calculated allowable concentration of chlordane in EFPC surface water is:

100 μg/kg / 37,800 L/kg = 0.0026 μg/L

This value is 60% of the value recommended by Eisler (1990). A PRG equal to the FWS recommendation would most likely protect piscivorous birds but might not protect all predatory fish.

PAHs. Evaluation of the potential toxicity of PAHs to piscivorous predators (Sect. 6.4.1.1) led to the conclusion that dietary exposures to PAHs in EFPC are not currently of concern. Projected body burdens calculated by use of BCFs and ARAR concentrations of PAHs (Table 6.73) were higher than PAH body burdens observed in the EFPC aquatic biota samples. Therefore, ARARs for PAHs in surface water are adequately protective for piscivorous predators.

7.3.3.5 Evaluation of PRGs for surface water

An evaluation of PRGs calculated for surface water and the criteria used to calculate them is presented in Table 7.10. Comparing ARARs to risk-based PRGs shows that the AWQCs for all analytes evaluated are probably adequately protective of biota in the EFPC environment. PRGs for PCBs in surface water are shown in Fig. 7.3. All of the proposed PRGs are below the contract-required detection limit of 1 μg/L. Bioconcentration of PCBs results in accumulation of analytes in biota to detectable limits even when they are undetectable in water, and the concentration in water can be estimated from the observed body burdens in aquatic biota. Therefore, attainment of PRGs can be measured only by continued monitoring of aquatic biota.

7.3.4 Groundwater

Deep groundwater is not considered a major exposure pathway for ecological receptors. Because of lateral migration of groundwater from the EFPC floodplain to the creek, shallow groundwater is a major pathway, to terrestrial insects and worms by dermal contact and ingestion, and to plants by root uptake (Fig. 6.39). However, shallow groundwater is transient, and contaminants found there are presumed to come either by leaching from EFPC floodplain soils or from sources outside the EFPC floodplain. Treatability studies have shown that the concentration of mercury in a water leachate of EFPC floodplain soils was approximately 0.08% of the concentration in soil. This means that mobility of soil mercury in groundwater is minor. In addition, cleanup of groundwater would not remediate the sources of contaminants to the groundwater and would not be an effective remedy. Therefore, no remedial goals for ecological risk are being proposed for shallow groundwater.

7.3.5 Sediment

Instream sediments may be an important source of ecological exposure to contaminants released from the Y-12 Plant (Fig. 6.38) or from the EFPC floodplain (Fig. 6.39). Benthic infaunal invertebrates live within the sediment layer and are exposed by dermal absorption and

Table 7.10. Criteria and potential PRGs for surface water in EFPC

Criterion	Chemical	PRG (μg/L)	Remarks
AWQC	Mercury	0.012	ARAR, BDL[a]
	Cadmium	1.0	ARAR, BDL
	PCB	0.001	ARAR for total PCB, BDL
	Chlordane	0.0043	Not ARAR, recommended maximum, BDL
	PAH	0.03 to 0.1	Chemical-specific ARARs
FDA Action Limit	Mercury	0.09 to 0.34	Assumes bioconcentration; met by ARAR
	Cadmium	—	No FDA action limit established
	PCB	0.02	Assumes bioconcentration; met by ARAR
	Chlordane	0.008	Assumes bioconcentration; met by recommended limit of 0.0043 μg/L
	PAH	--	No FDA action limit established
Protection from toxicity	Mercury	0.03 to 0.45	Assumes bioconcentration; met by ARAR
	Cadmium	1.0	ARAR is protective
	PCB	0.004	Assumes bioconcentration; met by ARAR
	Chlordane	0.2 to 3	Met by recommended limit of 0.0043 μg/L
	PAH	5	ARAR is protective
Protection of predators	Mercury	0.0045 to 0.022	Valid only if surface water is the direct source of contaminant uptake. Could be modified by further food chain modeling.
	PCB	0.005 to 0.03	
	Chlordane	0.0026	
	PAH	—	ARAR is protective

[a] Below Detection Limits

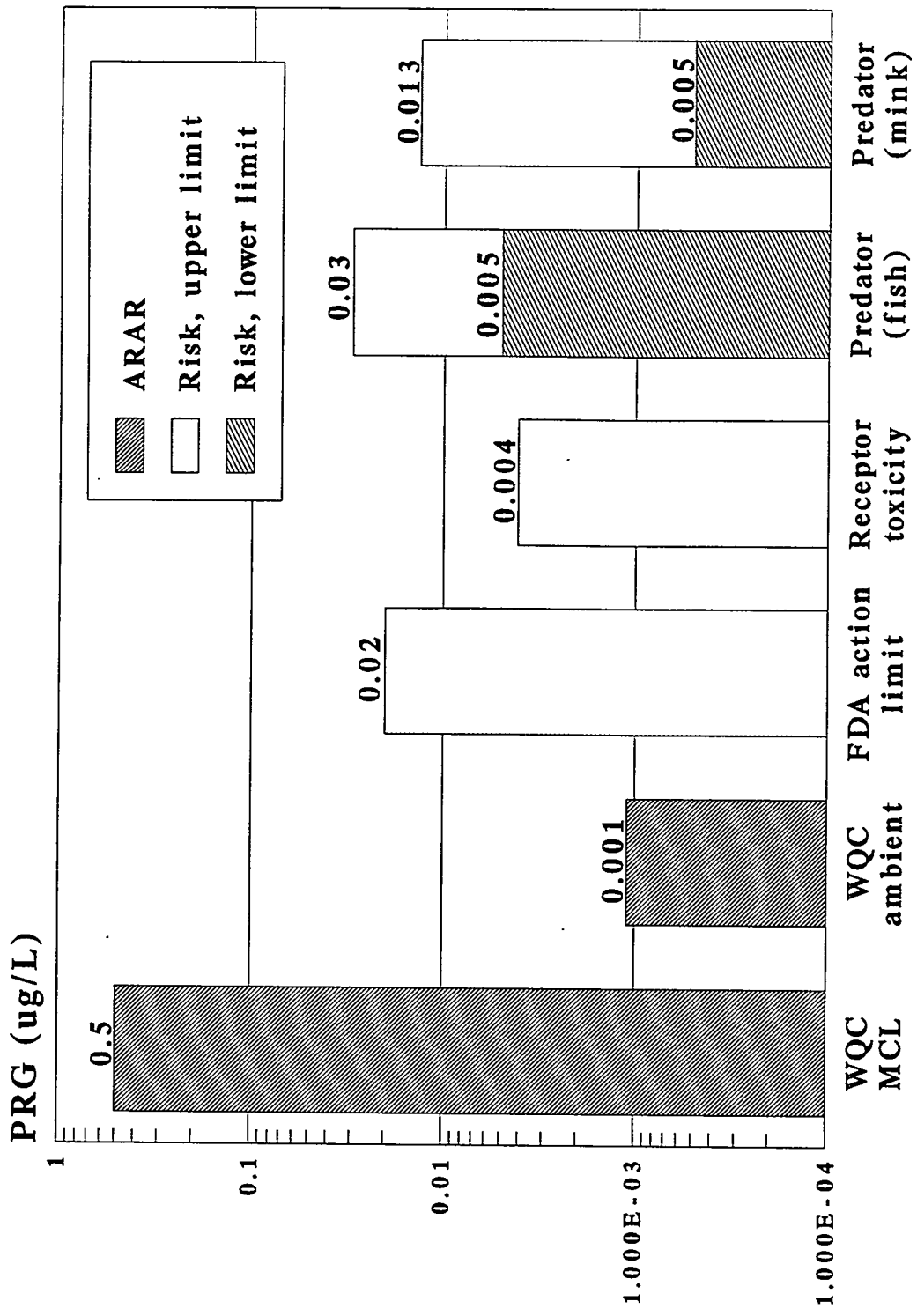

Fig. 7.3. Comparison of PRGs for PCBs in EFPC surface water.

ingestion, and crayfish and stonerollers may be exposed by incidental ingestion of sediment during feeding. Sediment may also provide the means for exposure at other levels of the aquatic food chain. Advisory action levels for sediment concentrations have been proposed by NOAA. These values are not ARARs, but are TBC guidance intended to identify sediments for which further study may be necessary.

A number of methods have been proposed for the assessment of sediment toxicity (Adams et al. 1992). They include the equilibrium partitioning (EP) method, the adverse effects threshold (AET) method, and other effects-based methods. These methods will be discussed in the following subsections. The methods can be used to derive sediment quality criteria (SQCs) based on the toxicological properties of the sediment or on considerations of further transport pathways and exposure routes.

7.3.5.1 NOAA action levels for sediment

The proposed effect levels for mercury, PCBs, and total PAHs in sediment (mg/kg) are:

	Mercury	Cadmium	PCB	PAH
Low effects range:	0.15	5	0.05	4
Median effects range:	1.3	9	0.4	35
Overall AET:	1	5	0.37	22

These values are intended as trigger values to initiate assessment of sediment to determine whether cleanup is required. The overall AET of 1 mg/kg for mercury is exceeded in most of the sediment samples analyzed for the EFPC RI; 1 sediment composite exceeded the median effects range and 3 exceeded the low effects range for PCBs, and 1 sediment composite exceeded the low effects range for PAHs. Therefore, according to the intended use of NOAA sediment action levels, it is appropriate to make additional assessments of risks from instream sediments. Accordingly, evaluation of potential exposures to sediment and establishment of PRGs for sediment are presented in the following subsections.

7.3.5.2 Equilibrium partitioning

The EP method assumes that the most important exposure to sediment contaminants is via pore water, and that contaminant concentrations in pore water and in the particulate or solid phase come to a chemical equilibrium. Therefore, the concentration of contaminant in one phase can

be predicted if the concentration in the other phase and the equilibrium coefficient are known. The advantage of this method is that partition coefficients can be predicted from octanol-water partition coefficients (K_{ow}) for many organic compounds, so site-specific data are not required. The disadvantages are (1) that concentrations of organic compounds in water may be below detection limits but still exceed AWQC, (2) site-specific data are required to calculate the partition coefficient for ions and polar organic compounds, and (3) partition coefficients are only useful to describe pore water in equilibrium with sediment. EPA may be moving toward the use of EP to develop SQCs (Adams et al. 1992).

Mercury. The binding of ions and other polar compounds to sediments is described by a partition coefficient, K_D, which describes the ratio of concentrations of a chemical in solid and liquid phases (e.g., SQC and AWQC, respectively). Using K_D and a water quality criterion (AWQC), the corresponding SQC can be derived:

$$SQC = AWQC \times K_D$$

K_D is specific for both the compound and the chemical nature of the sediment, and it is not always possible to derive. However, values of K_D for the binding of mercury to EFPC sediments were calculated from results of a mesocosm study reported by Turner et al. (Appendix Q). Using the AWQC for protection of freshwater biota of 0.012 μg/L (12×10^{-6} mg/L) and the mean K_D value for total mercury from the mesocosm study (8.85×10^5 L/kg), the calculated SQC for mercury is:

$$SQC = 12\times10^{-6} \text{ mg/L} \times 8.85\times10^5 \text{ L/kg} = 10.6 \text{ mg/kg}.$$

If particulate and soluble mercury were in equilibrium, the PRG for mercury in sediment would, therefore, be set at 10 mg/kg. However, the majority of EFPC sediments are coarse and their pore water is thought to be rapidly exchanged with surface water (Lee Wan 1991), so contaminants in pore water are not likely to be in equilibrium with sediment. Therefore, the PRG of 10 mg mercury/kg sediment is conservative.

PCBs. Nonpolar organic compounds bind to the organic carbon portion of soils and sediments, with an avidity (described by the constant K_{oc}) that is related to the total organic carbon (TOC) content of the particulates. Thus, a site-specific K_D is not necessary to calculate equilibrium partitioning for organic compounds when TOC is specified. The relationship used is the identity:

$$K_D = K_{oc} \times f_{oc}$$

where f_{oc} is the fractional organic carbon content of the particulates.

Because $SQC = AWQC \times K_D = AWQC \times K_{oc} \times f_{oc}$

and

$$SQC_{oc} = AWQC \times K_{oc}$$

it follows that

$$SQC = SQC_{oc} \times f_{oc}$$

For PCBs, AWQC = 0.001 μg/l = 1.0×10^{-6} mg/L

$K_{oc} = 7.8 \times 10^5$ L/kg (Lyman et al. 1990)

and

$$SQC_{oc} = 1.0 \times 10^{-6} \text{ mg/L} \times 7.8 \times 10^5 \text{ L/kg}$$
$$= 0.78 \text{ mg/kg} = 780 \ \mu\text{g/kg}$$

This result must be corrected for TOC. Values for TOC in EFPC sediments are not available, but soil TOC in the EFPC floodplain (a potential source of instream sediments) ranged from 1% to 11%. The most likely exposure (MLE) is assumed to be the mean exposure value and the reasonable maximum exposure (RME) is either the 95% upper confidence limit of mean of the exposure distribution or the maximum observed value, whichever is lower. For the calculation of sediment PRGs, the 95% lower confidence limit of the mean TOC was used because exposure is inversely related to sediment TOC. The mean TOC for upper horizon soils was 3.33%, with a 95% lower confidence limit of 2.15%. Using this range for EFPC sediments,

SQC = 26 μg/kg (MLE) to 17 μg/kg (RME)

Sediments used in the mesocosm study by Turner et al. (Appendix Q) had a range of 6.1 to 7.5% carbon, which computes to a PRG range of 47.5 to 58.5 μg/kg for these sediments. Therefore, a conservative sediment PRG for PCBs is 17 μg/kg.

Chlordane. The sediment PRG for chlordane is calculated similarly. The AWQC for chlordane is 4.3×10^{-6} mg/L and $K_{oc} = 1\times10^{6}$ L/kg. Therefore,

$$SQC_{oc} = 4.3\times10^{-6} \text{ mg/L} \times 1\times10^{6} \text{ L/kg}$$
$$= 4.3 \text{ mg/kg}$$

and for the TOC range of EFPC floodplain soils,

$$SQC = SQC_{oc} \times TOC = 143 \ \mu\text{g/kg (MLE) to } 90 \ \mu\text{g/kg (RME)}$$

For the mesocosm sediments (Appendix Q), the calculated SQC range is 262 to 322 μg/kg. Therefore, a conservative PRG for chlordane in sediment of 90 μg/kg is indicated.

PAHs. K_{oc}s for PAHs are not directly available. However, K_{oc} can be calculated from K_{ow} (Lyman et al. 1990) by using the empirical relationship

$$\log K_{oc} = \log K_{ow} - 0.21$$

[10 data points, $r^2 = 1.0$ (Karickhoff et al. 1979)]. Using the resulting values of K_{ow} for selected PAHs, along with the observed range of TOC for EFPC floodplain soils, sediment PRGs were derived to protect surface water at levels that are ARARs for drinking water or for recreational contact, whichever is lower (Table 4.1). The calculated values are given in Table 7.11.

7.3.5.3 Adverse Effects Threshold

The AET approach assumes that individual toxicants can be identified as sole causes of specific toxic responses in exposed biota. Therefore, the sediment cannot contain multiple chemicals with similar or joint modes of action, which is probably not true of EFPC sediments. The AET is calculated by assembling site-specific information on the adverse biological effects of exposure to sediment, i.e., population structure, species richness and diversity, numbers of individuals, contaminant body burden, and any other available relevant ecological parameters. The results are compared to data from one or more reference sites to identify sites where adverse

Table 7.11. Calculation of PRGs for PAHs in EFPC sediments

Compound	ARAR (μg/L)		log K_{ow}	Calculated K_{oc} (L/kg)	PRG for sediment (mg/kg)
	Domestic water supply (MCL)	Recreation			
Benzo[a]anthracene	0.1	0.3	5.61	2.57×10^5	230-2600
Benzo[a]pyrene	0.2	0.3	6.06	7.08×10^5	1250-14,000
Benzo[b]fluoranthene	0.2	—[a]	6.06	7.08×10^5	1250-14,000
Benzo[k]fluoranthene	0.2	—	6.06	7.08×10^5	1250-14,000
Chrysene	0.2	0.03	5.61	2.57×10^5	70-770
Dibenzo[a,h]anthracene	0.3	—	6.77	3.63×10^6	9900-108,000
Pyrene	—	0.03	4.88	4.68×10^4	12.5-140

[a]No ARAR promulgated.

impacts are perceived to occur. Sediment contaminant concentrations are evaluated to determine the lowest contaminant concentration at sites that show adverse effects. This concentration is chosen as the AET, and all sediments are cleaned up to that level. The advantage of this method is that it is site-specific. The disadvantages are that it requires data from an unimpacted reference site, it does not identify the chemicals causing the adverse effects, and it does not have much predictive power.

The effects typically used in the AET assessment are population measures for benthic invertebrates. The EFPC RI report states that nearly every measure of effect to benthic populations in EFPC showed an impact at every sampling site relative to the Hinds Creek reference site. Thus, there is no sediment concentration that is not associated with adverse effects, and use of the AET method would indicate that all sediments in EFPC should be removed. However, because it was concluded elsewhere in the RI report (Sect. 6.4.1.2) that the release of contaminants from the Y-12 Plant to surface water is the primary exposure route for aquatic biota, it is not reasonable to conclude that all of the impacts to benthic invertebrates are a result of sediment contamination and further, that all instream sediments should be cleaned up to the concentrations observed at Hinds Creek.

Observations by the BMAP have demonstrated partial ecological recovery of EFPC in response to contaminant control and cleanup activities at the Y-12 Plant (Loar 1992). Upon completion of cleanup of Y-12 Plant contaminant sources, aquatic biota in EFPC should equilibrate with sediment or floodplain soil sources. At that time, the AET method should be reconsidered.

7.3.5.4 Other effects-based methods

Other effects-based methods also involve comparison of sediment contaminant concentrations with tissue body burdens, community structure, and measured sediment toxicity in different combinations. Weight-of-evidence considerations are then used to conclude what sediment cleanup levels are required. These methods assume that sediment contaminants are the principal source of toxicity to the aquatic biota being evaluated. In contrast, the aquatic biota in the surface water column of EFPC appear to be impacted mainly by surface water (Sect. 6.4.1.2). Therefore, these methods cannot be applied at the present time. They should be reconsidered after releases from the Y-12 Plant have been controlled.

7.3.5.5 Evaluation of ecologically-based PRGs for sediment

The potential PRGs for sediment are listed in Table 7.12. Mercury concentrations in 8 of 27 sediment composites taken during Phase Ib sampling exceeded the PRG of 10 mg/kg. PCB concentrations in sediment composites ranged from undetectable to a maximum of 420 μg/kg, which exceeded the PRG range for all soils and sediments tested.

Exposure to contaminants in sediment is assumed to result from transport of contaminants into water trapped in interstitial pores of the sediment, so the nature of the pore water is very important in determining exposure. Fine-grained sediment, which may be found in pools and other areas with low flow rates, have more pores than coarse-grained sediments. Pore water in fine sediments also exchanges with surface water more slowly than pore water in coarse sediment, so pore water contaminant concentrations can build up to higher levels in fine sediments. EFPC sediments are mostly coarse because the high flow rate of the creek removes the fine material. Pore water in coarse sediments is more likely to be similar to surface water, and it was assumed in the RI that sediment pore water contaminants were the same as those in surface water.

Surface water, which received mercury loading from the Y-12 Plant, is currently the major source of mercury contamination to aquatic biota. The average sediment mercury level in the sediment composite representing Site 1 was 18 mg/kg (Fig. 6.43). At this site, the observed mercury concentration in surface water was 0.54 μg/L, whereas the calculated equilibrium concentration would be approximately 18,000 μg/kg / 8.5×10^5 L/kg = 0.02 μg/L, lower than the observed value by a factor of approximately 25. This implies that sediment could not supply the observed concentration of mercury. Therefore, cleanup levels should be applied to instream sediments only after the Y-12 Plant surface water source has been remediated.

7.3.6 Soil

Terrestrial animals were shown to have accumulated concentrations of mercury exceeding guidelines for protection from toxicity and for protection of predators (Sect. 6.4.1.1). Soil is a potential source of exposure by direct ingestion and inhalation and indirectly through the food chain. These pathways will be discussed in the following subsections.

Table 7.12. Criteria and potential PRGs for sediment in EFPC

Criterion	Chemical	PRG (mg/kg sediment)	Remarks
NOAA Action Level	Mercury	1.0	TBC, advisory only; triggers investigation
	Cadmium	5.0	TBC, advisory only; triggers investigation
	PCB	0.37	TBC, advisory only; triggers investigation
	Chlordane	--	Does not exist for chlordane
	PAH	22	Chemical-specific. See Table 7.11
Equilibrium partitioning	Mercury	10	Best available, but system is not currently at equilibrium
	Cadmium	--	Site-specific K_D not available
	PCB	0.017 to 0.026	Best available, but system is not currently at equilibrium
	Chlordane	0.09 to 0.143	Best available, but system is not currently at equilibrium
	PAH	12.5 to 108,000	Chemical-specific. See Table 7.11

7.3.6.1 Ingestion

Exposure by ingestion of soil is calculated by modifying the standard formula for calculating the hazard quotient (EPA 1989):

$$EQ = (C_{soil} \times 10^{-6}\ kg/mg \times SI \times FI \times ABS)/(CD \times BW \times 10^{-3}\ kg/g) \text{ and}$$

$$C_{soil} = (EQ \times CD \times BW \times 10^{-3})/(SI \times 10^{-6} \times FI \times ABS)$$

where

C_{soil} =	Contaminant concentration in soil (mg contaminant/kg soil)
EQ =	Exposure quotient = 1
CD =	Comparison dose (mg contaminant/kg body weight/day)
BW =	Body weight (25 g)
SI =	Soil intake (100 mg soil/day)
FI =	Fraction of soil from contaminated area (1.0)
ABS =	Fraction of ingested dose absorbed (1.0)

Justification and comments on variables:

EQ: The target exposure quotient is always 1. Assuming other parameters are known reliably, a higher value would not be protective, and lower values would be overly protective.

CD: The comparison dose is the toxicological benchmark to which the organism is to be protected. It should preferably be a Lowest Observable Effects Level, No Observed Adverse Effects Level, or similar measure of effect.

BW: Body weight of shrews is typically approximately 25-40 g. Variability in body weight is likely to be compensated by a similar variability in soil ingestion.

SI: Incidental ingestion of soil by small mammals is likely to be variable, depending on feeding habits. Birds may ingest soil intentionally for grit to help grind their food. For this calculation, an arbitrary value of 100 mg/day (approx. 0.4% of body weight) is used. This

value is probably overly conservative for mice, but it may be an underestimate for shrews, which burrow and forage in the soil.

FI: It is assumed here that the home range of the subject animal is restricted to the contaminated area of the EFPC floodplain (FI = 1). That assumption is probably reasonable for mice and shrews.

ABS: The absorption factor may depend on both the contaminant and the medium in which the contaminant is contained. Typically, the absorption factor of organic chemicals is assumed to be 1, whereas it is usually lower for inorganic chemicals. If the medium used to determine the comparison dose was the same as the exposure medium, no correction for absorption should be made. Toxicological studies of inorganic chemicals may be based on absorption of 20% or less of the administered dose (EPA 1989). Feeding studies have shown that absorption of mercury by laboratory mice from contaminated soil taken from the EFPC floodplain was less than 10% (Revis et al. 1989). However, for the calculation of PRGs, a conservative value of ABS = 1 is used. As a result, the PRG for mercury may be conservative by a factor of approximately 2 to 4.

Mercury. Based on the laboratory study by Revis et al. (1989), incidental ingestion of mercury in EFPC floodplain soil is not expected to cause toxic effects to mammals. Evaluation of mercury ingestion with soil follows:

A dietary concentration of 100-200 mg Hg/kg/day caused histological damage to rat kidney, whereas no damage was observed in mink fed 10 mg/kg/day (Table 6.42). Revis et al. (1989) reported a NOEL of 13.7 mg/kg for mice fed soil-bound mercury. For this derivation, the range of these numbers, 13.7-100 mg/kg/day, will be used as values for CD.

Calculation of PRG: with the values listed above,

$$C_{soil} = (1 \times 13.7 \text{ to } 100 \times 25 \times 10^{-3})/(100 \times 10^{-6} \times 1 \times 1)$$
$$= 3420\text{-}25{,}000 \text{ mg/kg}.$$

Because mercury concentrations above 1600 mg/kg were not observed in surface soils, it is concluded that the soil ingestion pathway is not of major significance for mercury exposure of small mammals.

Cadmium. The maximum concentration of cadmium observed in soil during Phase Ia sampling was 41.3 mg/kg (Table 3.32). In the study by Revis et al. (1989) no evidence was found for histological damage to liver or kidney, depression of reproduction, or alterations in behavior patterns in mice fed soil containing up to 93 mg/kg cadmium. Inasmuch as the diet contained 5% soil (Revis et al. 1989), it is unlikely that a higher exposure to cadmium would be encountered by small mammals in the EFPC floodplain. Therefore, a PRG of 90 mg/kg is recommended for cadmium in soil.

PCBs. Rats showed no measurable effects at dietary levels of PCB equivalent to 0.5 mg/kg BW daily (Eisler 1986). Using a safety factor of 100, a tolerable exposure limit of 5.0 μg/kg PCB BW daily was proposed by FWS (Eisler 1986). This value (CD = 0.005 mg/kg) was used for the calculation of exposure for small mammals.

Calculation of PRG:

$$C_{soil} = (1 \times 0.005 \times 25 \times 10^{-3})/(100 \times 10^{-6} \times 1 \times 1)$$
$$= 1.25 \text{ mg PCB/kg.}$$

Therefore, the recommended PRG for PCBs in soil is 1.25 mg/kg.

Chlordane. A comparison dose of 0.1 to 0.3 mg/kg for birds and 5 mg/kg for rats was used to calculate the amount of chlordane ingested through soil.

Calculation of PRG: based on these values,

$$C_{soil} = (1 \times 0.1 \text{ to } 5 \times 25 \times 10^{-3})/(100 \times 10^{-6} \times 1 \times 1)$$
$$= 25 \text{ to } 1250 \text{ mg/kg.}$$

The highest concentrations of alpha-chlordane and gamma-chlordane in EFPC floodplain soil were 24 μg/kg and 8.3 μg/kg, respectively (Table 3.32). Therefore, remediation of soil for chlordane contamination appears not to be necessary to protect birds and small mammals from chlordane toxicity by soil ingestion.

PAHs. A comparison dose of 30 mg/kg for rats was used to calculate the amount of PAHs ingested through soil (Eisler 1987b).

Calculation of PRG based on this value:

$$C_{soil} = (1 \times 30 \times 25 \times 10^{-3})/(100 \times 10^{-6} \times 1 \times 1)$$
$$= 7500 \text{ mg/kg.}$$

The highest concentrations of PAHs in EFPC floodplain soil were below 10 mg/kg (Table 3.32). Therefore, remediation of soil for PAH contamination is not necessary to protect birds and small mammals from toxicity by soil ingestion.

7.3.6.2 Inhalation

Animals living close to or in the ground may be exposed to volatile chemicals in the soil. PRGs for these chemicals in soil are addressed as soil PRGs because cleanup of soil would be required to reduce any deleterious inhalation exposures.

The allowable soil concentration of a volatile compound transported to air may be calculated (EPA 1991b) by the formula:

$$C_{soil} = (EQ \times BW \times AT \times 365 \text{ days/yr})/[(ED \times EF \times IR_{air})/(RfD_{air} \times VF)]$$

where:

C_{soil} = Contaminant concentration in soil (mg contaminant/kg soil)
EQ = Exposure quotient = 1
BW = Body weight (25 g)
AT = Averaging time (2 yr)
ED = Exposure duration (2 yr)
EF = Exposure frequency (365 days/yr)
IR_{air} = Inhalation rate (0.5 m^3/day, arbitrary assumption based on data for other small mammals)
RfD_{air} = Inhalation chronic reference dose
VF = Volatilization factor (m^3/kg)

$$VF = [(LS \times V \times DH)/A] \times [(\pi \times \alpha \times T)^{1/2}/(2 \times D_{ei} \times E \times K_{as} \times 10^{-3} \text{ kg/g})]$$

where:

LS = Length of side of area (100 m)
V = Wind speed in mixing zone (default 2.25 m/sec)
DH = Diffusion height (0.01 m, arbitrary 10-cm fossorial exposure zone)
A = Area (1 hectare = 1×10^8 cm^2 in ecological sampling site)
$\pi \approx$ 3.1416
α = $(D_{ei} \times E)/[E + p_s(1-E)/K_{as}]$
  D_{ei} = Effective diffusivity ($D_i \times E^{0.33}$)
  D_i = Molecular diffusivity (chemical-specific)
  E = Soil porosity (default 0.35)
  p_s = Soil density (default 2.65 g/cm^3)
  K_{as} = Soil/air partition coefficient = $(H \times 41)/K_D$
    H = Henry's law constant (chemical-specific)
    K_D = Soil-water partition coefficient (chemical-specific)
T = Exposure duration (6.3×10^7 sec = 2 yr)

Chemical-specific values used in this derivation are listed in Table 7.13.

Volatilization of chemicals from soil depends on the soil-water partition coefficient, K_D, which for organic chemicals is related to the TOC. A range of exposures based on the range of TOC in soil was used in the following calculations for organic chemicals. The mean value of TOC in the upper horizon soils was used as the MLE value. The RME is usually taken as the 95% upper confidence limit of the mean exposure concentration. Since volatilization is inversely proportional to K_D, the RME for volatiles in soil is calculated in the following subsections by using the 95% lower confidence interval of TOC. The mean concentration of TOC in the uppermost horizon of EFPC floodplain soil was 3.3%, with a 95% lower confidence limit of 2.2%. These values were used to calculate MLE and RME exposures, respectively.

Mercury. The predominant form of mercury in EFPC floodplain soils is a firmly bound, non-volatile form associated with particulates (Appendix G). The majority is in an ionic form, perhaps including sulfides or complexes with organic material of plant origin. Mercury vapors may be released from soil if elemental mercury is present. Surveillance of air close to EFPC floodplain soils showed that ambient concentrations of mercury vapors were no different from those at a background reference location (Sect. 3.2.8; Appendix H). Organic mercury comprises a small fraction of the total soil mercury. Methyl-mercury was shown to be present in soil at an average of <0.01% of the total mercury concentration (Appendix G), and dimethyl mercury is

Table 7.13. Chemical-specific parameters for inhalation exposure to contaminants in EFPC floodplain soil

Chemical	Parameter			
	RfD	D_i	H	K_D
	Inhalation reference dose	Molecular diffusivity	Henry's law constant	Soil-water partition coefficient
Mercury	13.7 to 100 mg/kg/day	0.12 cm^2/sec (EPA 1988)	0.011 atm-m^3/mol (Lyman et al. 1990)	8.85×10^5 cc/g (Turner et al. 1992)
PCB	0.5 mg/kg/day	0.056 cm^2/sec (Lyman et al. 1990)	0.0071 atm-m^3/mol (Lyman et al. 1990)	$K_{oc} \times Oc = 7.8 \times 10^5 \times$ (0.01 to 0.11)
Chlordane	1.25 mg/kg/day	0.1187 cm^2/sec (Lyman et al. 1990)	0.00005 atm-m^3/mol (Lyman et al. 1990)	$K_{oc} \times Oc = 1 \times 10^6 \times$ (0.01 to 0.11)

typically present in environmental media at only a small fraction of the concentration of methyl-mercury. Therefore, volatile organic mercury should not present a significant exposure to biota.

The calculated value of C_{soil} for mercury was 185-1350 mg/kg, depending on the values of the chronic reference dose used. This value assumes that all soil mercury is in the firmly bound ionic form. Because the majority of the mercury is actually ionic or bound in a non-volatile form, the lower PRG of 185 mg/kg for inhalation of vapors is probably adequately conservative and is therefore recommended as the PRG for inhalation of mercury vapors from soil.

Cadmium. Cadmium salts are not significantly volatile. Therefore, no PRG for inhalation of cadmium from soil was calculated.

PCBs. Using the range of TOC values given above (Sect. 6.2), the calculated values of C_{soil} for PCB ranged from 2.1 mg/kg for the MLE to 1.7 mg/kg for the RME. Therefore, a conservative PRG for inhalation of contaminants from soil is 1.7 mg/kg. The calculated values are above most PCB concentrations observed during Phase Ia sampling and analysis of soil, but are exceeded by the maximum observed concentrations in land use segments 1 and 4 (Table 3.32).

Chlordane. Similarly, the calculated values of C_{soil} for chlordane ranged from 39 mg/kg for the RME to 49 mg/kg for the MLE. The highest concentrations of alpha-chlordane and gamma-chlordane in EFPC floodplain soil were 24 μg/kg and 8.3 μg/kg, respectively (Table 3.32). Therefore, remediation of soil to protect against chlordane toxicity by inhalation appears not to be necessary.

PAHs. Chemical-specific data needed to calculate volatilization of PAHs were not all available, and some parameters were estimated by comparison to similar compounds (Table 7.13). Estimates of soil PRGs were made for three representative PAHs, pyrene (4 rings), benzo(a)pyrene (5 rings), and benzo(g,h,i)perylene (6 rings). The resulting PRGs for soil ranged from 28 mg/kg for the RME exposure to pyrene to 570 mg/kg for the MLE exposure to benzo(g,h,i)perylene. The highest concentrations of PAHs in EFPC floodplain soil were below 10 mg/kg (Table 3.32). Therefore, remediation of soil to protect against PAH toxicity by inhalation appears not to be necessary.

7.3.6.3 Protection of low-level predators in the EFPC floodplain from toxicity via the food chain

Contaminants are expected to enter the food chain of terrestrial biota by uptake from soil into plants, earthworms, and insects, as well as by the ingestion and possibly inhalation pathways described in subsections 7.3.6.1 and 7.3.6.2 above. Small mammals were shown to have body burdens of some contaminants above concentrations considered to be potentially toxic (Tables 6.42, 6.74; Eisler 1986, 1987a). The major route of contaminant accumulation is probably the food chain. Because elevated body burdens were observed in shrews and wrens but generally not in mice and voles, it appears that a diet of soil-dwelling arthropods and worms is a likely source for bioaccumulation of contaminants.

Although a quantitative relationship between soil contaminant concentration and body burden in terrestrial small mammals at the EFPC floodplain has not yet been demonstrated, food chain modeling data from other studies may be used to estimate potential PRGs based on toxicity. The model used (EPA 1987) assumes that contaminant body burdens in soil biota are proportional to the contaminant concentrations in the surrounding soil. If the ratio of body burden to soil concentration [bioaccumulation factor (BAF)] is known for a given organism and contaminant, the body burden in the receptor can be calculated from the average soil concentration. Similarly, if there is a target body burden thought to be safe to the receptor or its predators, the corresponding soil concentration can be calculated.

Mercury. In a study of mercury accumulation from soil by earthworms, a BAF of 0.34 mg mercury/kg (dry weight) (mg mercury/kg soil)$^{-1}$ was calculated (EPA 1985a). The water content of earthworms has been reported to range from 70% to 95% (Minnich 1977). Using the median water content of 82.5%, a fresh-weight basis BAF was calculated:

$$\begin{aligned} \text{BAF} &= 0.34 \text{ mg/kg dry wt. (mg/kg soil)}^{-1} \times 0.175 \text{ dry wt./fresh wt.} \\ &= 0.06 \text{ mg/kg fresh wt. (mg/kg soil)}^{-1} \end{aligned}$$

The target daily dietary intake range for protection of small mammals from toxicity to mercury is 13.7 (Revis et al. 1989) to 100 mg/kg (Table 6.42). Using the BAF for earthworms,

$$\begin{aligned} C_{soil} &= 13.7 \text{ to } 100 \text{ mg/kg} / 0.06 \text{ mg/kg (mg/kg soil)}^{-1} \\ &= 228 \text{ to } 1670 \text{ mg/kg soil} \end{aligned}$$

The lower value reflects a no-effect level, whereas the higher value represents reported damage to organs of the receptor. Therefore, these values represent a range below which there is no concern and above which there is a threat to low-level predators. On the basis of this calculation, the proposed soil PRG for protection of predators of earthworms (i.e., small mammals and birds) from toxicity of mercury is 230 mg/kg.

Cadmium. A BAF of 13.7 mg cadmium/kg (dry weight) (mg cadmium/kg soil)$^{-1}$ was calculated for uptake of cadmium from soil by earthworms (EPA 1985b). Using a median water content of 82.5%, a fresh-weight basis BAF was calculated:

BAF = 13.7 mg/kg dry wt (mg/kg soil)$^{-1}$ x 0.175 dry wt./fresh wt.
= 2.4 mg/kg fresh wt. (mg/kg soil)$^{-1}$

The target daily maximum dietary intake range for protection of small mammals from toxicity to cadmium is 100 μg/kg diet (Eisler 1985). Using the BAF for earthworms,

C_{soil} = 0.1 mg/kg / 2.4 mg/kg (mg/kg soil)$^{-1}$
= 0.04 mg/kg

Concentrations of cadmium in EFPC floodplain soils are as much as 1000-fold above this value (Table 3.32). Because there is no indication that potential predators of earthworms have accumulated high levels of cadmium (Table 6.30), this transfer model appears not to be a realistic description of exposure. Therefore, this PRG for cadmium in soil is rejected for EFPC floodplain soils, and it is concluded that cadmium in soils need not be remediated to protect low-level predators.

PCBs. Dietary exposures of low-level predators to PCBs could not be calculated because earthworms and aquatic insects, the major portion of their diets, were not analyzed for PCBs. If the mean PCB concentration in terrestrial insects represents the dietary exposure of wrens and shrews, the exposure quotient (calculated as in Table 6.81) would be approximately 0.3 for both. Therefore, current exposures are probably not a risk to low-level predators, and PRGs for soil remediation are not required.

Chlordane. The current risks to terrestrial biota from chlordane in soil are very low (Sect. 6.4.1.1). Therefore, PRGs for soil remediation are not required.

PAHs. The current risks to terrestrial biota from PAHs in soil are very low (Sect. 6.4.1.1). Therefore, PRGs for soil remediation are not required.

7.3.6.4 Protection of higher predators from contaminant toxicity via the food chain

Biomagnification increases the body burdens of predators over those of their prey. For example, the average concentration of Aroclor 1260 in terrestrial insects sampled in the EFPC floodplain was 210 μg/kg, while the average concentration in wrens, which prey on insects, was approximately ten-fold higher, and the average concentration in shrews was approximately five-fold higher. Mercury was not detected in four of the six floodplain samples of terrestrial insects but had accumulated in wrens (average 3.5 mg/kg) and shrews (average 4.9 mg/kg). PRGs must protect predators from bioaccumulation to toxic levels as a result of eating contaminated prey. Small mammals, especially shrews, must be considered as both predator and prey. The following paragraphs present soil PRGs for protection of top predators. The feeding ranges of top predators and the typical dietary mix (Sect. 6.4.1.1) are included in these derivations.

Mercury. Soil PRGs for the protection of low-level predators from mercury in the food chain were presented in Sect. 7.3.6.3. Because the observed exposure quotients for mercury were 7.0 in shrews and 4.2 in wrens (Table 6.81) and the highest calculated exposure quotient for top predators was 1.2 in owls, a reduction of mercury concentrations in soil to levels that are protective of shrews and owls should adequately protect top predators as well. Therefore, the recommended PRG for protection of top predators from mercury in the food chain is 230 mg/kg soil.

Cadmium. Cadmium was not detected in mice (Table 6.27) or vole (Table 6.30), nor in one of the two shrews sampled (Table 6.30). The cadmium body burden in the other shrew was less than three times the detection limit (Table 6.30). Therefore, it is unlikely that cadmium in prey animals represents a threat to predators. It is concluded that EFPC floodplain soils do not need to be remediated to protect top predators from cadmium exposure.

PCBs. The average PCB body burden in mice (Table 6.28) and vole (Table 6.30) from the EFPC floodplain was 0.20 mg/kg, whereas the average for shrews was 1.18 mg/kg (Table 6.30). Based on the predators' expected feeding ranges and dietary mixes (Table 6.80), the expected exposure of top predators to Aroclor 1260 was less than 0.04 mg/kg diet (Table 6.81). The combined body burden of PCBs in small mammals is therefore below the target value of

0.5 mg/kg for protection of terrestrial predators (Sect. 6.4.1.1), and remediation of PCBs in EFPC floodplain soils is not indicated for the protection of top predators.

Chlordane. The average alpha plus gamma chlordane body burden in mice (Table 6.29) and vole (Table 6.30) from the EFPC floodplain was 6.4 μg/kg, whereas the average for shrews was 17 μg/kg (Table 6.30). The combined body burden of chlordane in small mammals is therefore below the target value of 100 μg/kg diet for protection of terrestrial predators (Sect. 6.4.1.1). This implies that chlordane in prey taxa does not represent a threat to predators and that PRGs specifically intended to protect predators are not required.

PAHs. Reported acute oral LD_{50} values for PAHs are at least 50 mg/kg body weight (Eisler 1987). Allowable dietary concentrations would be derived by dividing by safety factors for chronic exposure and for an endpoint less severe than 50% lethality. Specific guidance for doing so is not provided by EPA, but it is unlikely that allowable dietary concentrations would be below 50 μg/kg, 1000-fold below the highest LD_{50} value.

The maximum body burden for any PAH observed in terrestrial biota was an estimated 53 μg acenaphthene/kg body weight in a mouse. Anthracene (maximum = 33 μg/kg in a mouse), benzo(a)pyrene (maximum = 12 μg/kg in terrestrial insects), and benzo(k)fluoranthene (maximum = 14 μg/kg in terrestrial insects) were observed in all samples; most PAH analytes were below detection limits in most of the samples of terrestrial biota (Tables 6.30 and 6.34). Therefore, it is concluded that PAHs in prey taxa do not represent a threat to predators and that remediation specifically intended to protect predators from PAHs in prey is not required.

7.3.6.5 Evaluation of soil PRGs

The potential PRGs for soil are listed in Table 7.14. Because no direct link has yet been established with the EFPC data between soil contaminant concentrations and body burdens in terrestrial receptors, PRGs for protection of predators from contaminants in soil are based on models of contaminant uptake. Contaminants other than mercury are not likely to pose a threat to higher predators.

Table 7.14. Criteria and potential PRGs for surface soil in the EFPC floodplain

Criterion	Chemical	PRG (mg/kg soil)	Remarks
Incidental ingestion of soil	Mercury	3240 to 25,000	Based on highly conservative estimates of ingestion
	Cadmium	90	
	PCB	1.25	
	Chlordane	25 to 1250	
	PAH	7500	
Inhalation of vapors from soil	Mercury	185 to 1350	Based on non-volatile mercury; elemental fraction is low
	PCB	1.7 to 2.1	Based on assumed range of TOC in soil
	Chlordane	39 to 49	
	PAH	28 to 570	
Food chain, protection from toxicity	Mercury	230 to 1670	Assumes proportional bioaccumulation in food chain
	Cadmium	—[a]	No apparent threat to low-level predators
	PCB	—	
	Chlordane	—	
	PAH	—	
Food chain, protection of predators	Mercury	230	Allows for mix of diet, range of predators
	Cadmium	—	No apparent threat to top predators
	PCB	—	
	Chlordane	—	
	PAH	—	

[a] Not derived because of lack of apparent threat to predators

7.4 COMPARISON OF PRGS FOR HUMAN HEALTH AND ECOLOGICAL PROTECTION

7.4.1 Introduction

PRGs for human health and for ecological protection are based on different exposure pathways, exposure concentrations, and receptor populations. As a result, PRGs are likely to be different for each environmental medium considered. Representative contaminants were evaluated for ecologically based PRGs, whereas a larger set of COCs was used for human health PRGs. The following subsection compares the PRGs for COCs evaluated for both human health (Sect. 7.2) and ecological risk (Sect. 7.3) in EFPC and EFPC floodplain media. The comparisons are summarized in Table 7.15.

7.4.2 Air

Air was not considered a significant exposure medium for either humans or ecological receptors. Therefore, remedial goals for air were not proposed for either human health or environmental protection.

7.4.3 Surface Water

The most important routes for exposure of humans to surface water is incidental ingestion and dermal contact during swimming and wading. In contrast, many aquatic biota are exposed continually by ingestion, by dermal contact and through the food chain. In general, PRGs for human exposure are much higher than for aquatic biota (Table 7.15).

The most conservative PRGs for aquatic biota are also ARARs, so they should be adopted. However, the attainment of many of them will have to be monitored by a continued surveillance of aquatic biota in EFPC. Because of current releases from the Y-12 Plant, PRGs for EFPC surface water are goals to be met after both cleanup of the EFPC floodplain and control of the releases from the Y-12 Plant. PRGs for mercury in surface water are shown graphically in Fig. 7.4. The ARAR for drinking water (MCL) is shown for comparison only, as surface water in EFPC is not a likely candidate for a drinking water source. The PRGs derived for human health (exposure by ingestion and dermal contact via the recreational use scenario) are also shown for comparison.

Table 7.15 Comparison of PRGs for protection of human health and the environment

Exposure medium	Receptor type	Chemicals				
		Mercury	Cadmium	PCBs	Pesticides	PAHs
Surface water (μg/L)	Human	1.28×10^{3}	—[a]	—	4.2-200[b]	0.03-0.1
	Ecological	0.012	1.0	0.001	0.004	0.03-0.1
Sediment (mg/kg)	Human	3.7×10^{4}	1.24×10^{5}	4.9	—	5-520
	Ecological	10	—	0.02-0.03	0.09-0.14	$12.5\text{-}1.08\times10^{5}$
Soil, ingestion (mg/kg)	Human	59; 253[c]	196; 845	0.1; 0.25	—	0.26-26
	Ecological	$3.2\times10^{3}\text{-}2.5\times10^{4}$ [d]	90	1.25	25-1250	7500
Soil, food chain (mg/kg)	Human	0.3; 0.25[e]	0.6; 0.5	0.003; 0.005	—	0.1-5.4; 0.004-1.2
	Ecological	230	—	—	—	—

[a]No PRG derived because current risks do not warrant remediation
[b]Range depends on specific compounds
[c]First number is for agricultural/residential exposure, second is for open land use
[d]Range depends on comparison dose
[e]First number is for produce pathway, second is for meat and dairy pathway

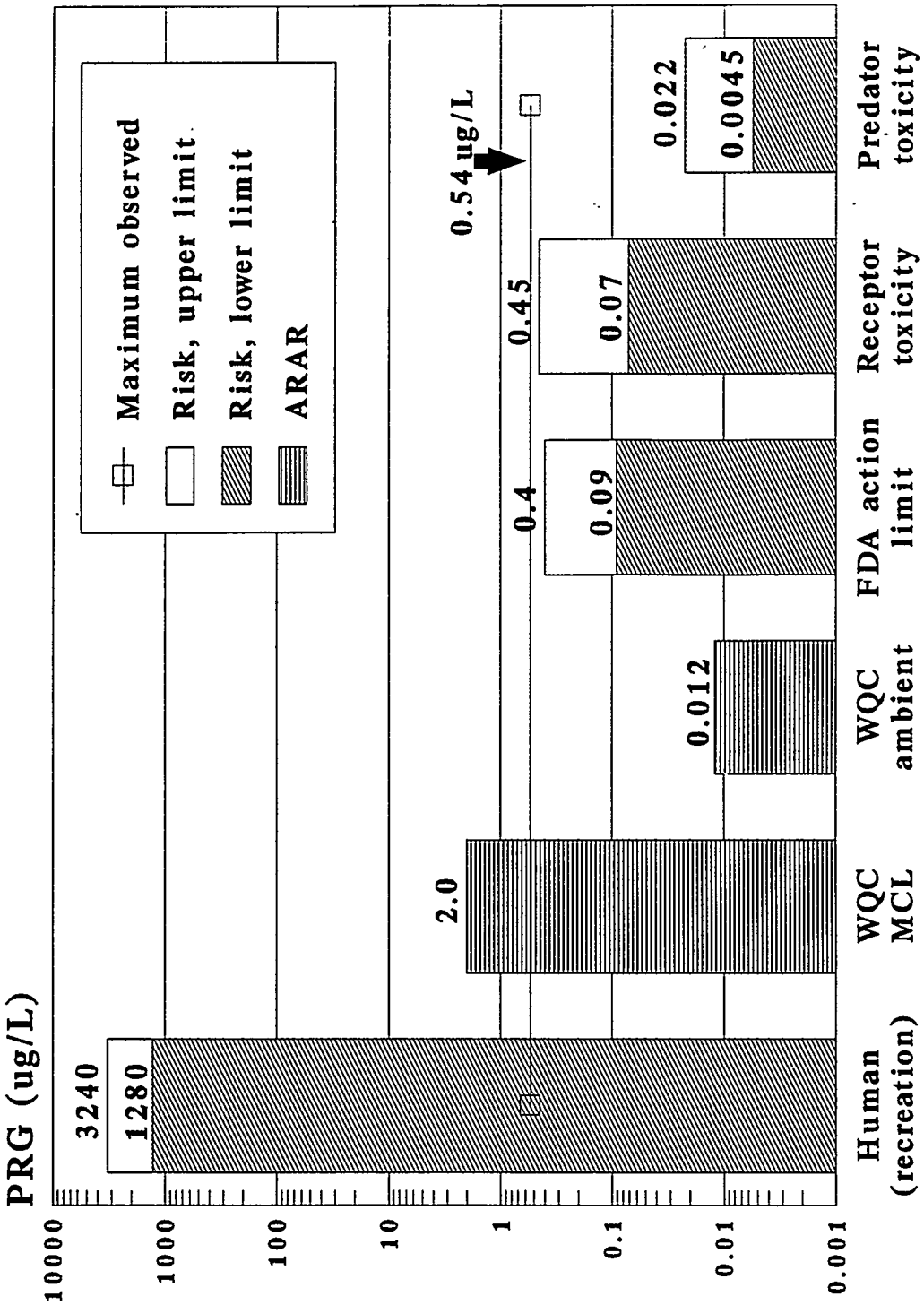

Fig. 7.4. Comparison of PRGs for mercury in EFPC surface water.

7.4.4 Groundwater

Groundwater is not an important current source of risk, and remediation is not required to protect humans or the environment. Potential future risks to humans drinking groundwater were used to derive groundwater PRGs (Table 7.7). Because samples were not taken from a suitable drinking water aquifer, these PRGs may not be appropriate for shallow groundwater remediation.

7.4.5 Sediment

Sediment exposure via direct contact during wading causes low risks to humans, and therefore, high PRGs (Table 7.15) in EFPC. In contrast, exposure of benthic invertebrates to sediment pore water may provide a major ecological risk, resulting in low PRGs for sediment. The methods for deriving ecologically based sediment PRGs have yet to be validated, so sediment PRGs especially should be further refined during the FS/EIS process. Figure 7.5 compares sediment PRGs for mercury and PCBs for protection of human health (recreational exposure), NOAA trigger levels for ecological protection, and values calculated by equilibrium partitioning.

7.4.6 Soil

PRGs for mercury in soil are shown in Fig. 7.6. Incidental ingestion of soil is not a major risk to either humans or ecological receptors in the EFPC floodplain. Therefore, PRGs for soil ingestion are high (Table 7.15). Because the mercury RfD for humans is much lower than the comparison doses used for ecological receptors, soil PRGs to protect humans from exposure to mercury in soil are lower than ecologically based PRGs for soil ingestion.

Exposure via the food chain resulted in lower PRGs than for soil ingestion for both human health and the environment (Table 7.15). It was recommended that human health-based PRGs for soil exposures to mercury by the food chain not be used in the FS because of uncertainty about published bioaccumulation factors from soil to plants and the low RfD for human ingestion of mercury. Mercury exposure to low-level predators via uptake from soil by earthworms was modeled to derive a soil PRG to protect those biota. The PRG derived in this way was approximately equal to the human health-based PRG for soil derived for commercial exposures.

Current exposures of top predators to PCBs, pesticides, and PAHs appear not to be high enough to require remediation. In contrast, because PCBs and PAHs are human carcinogens, their PRGs are much lower for humans than for ecological receptors.

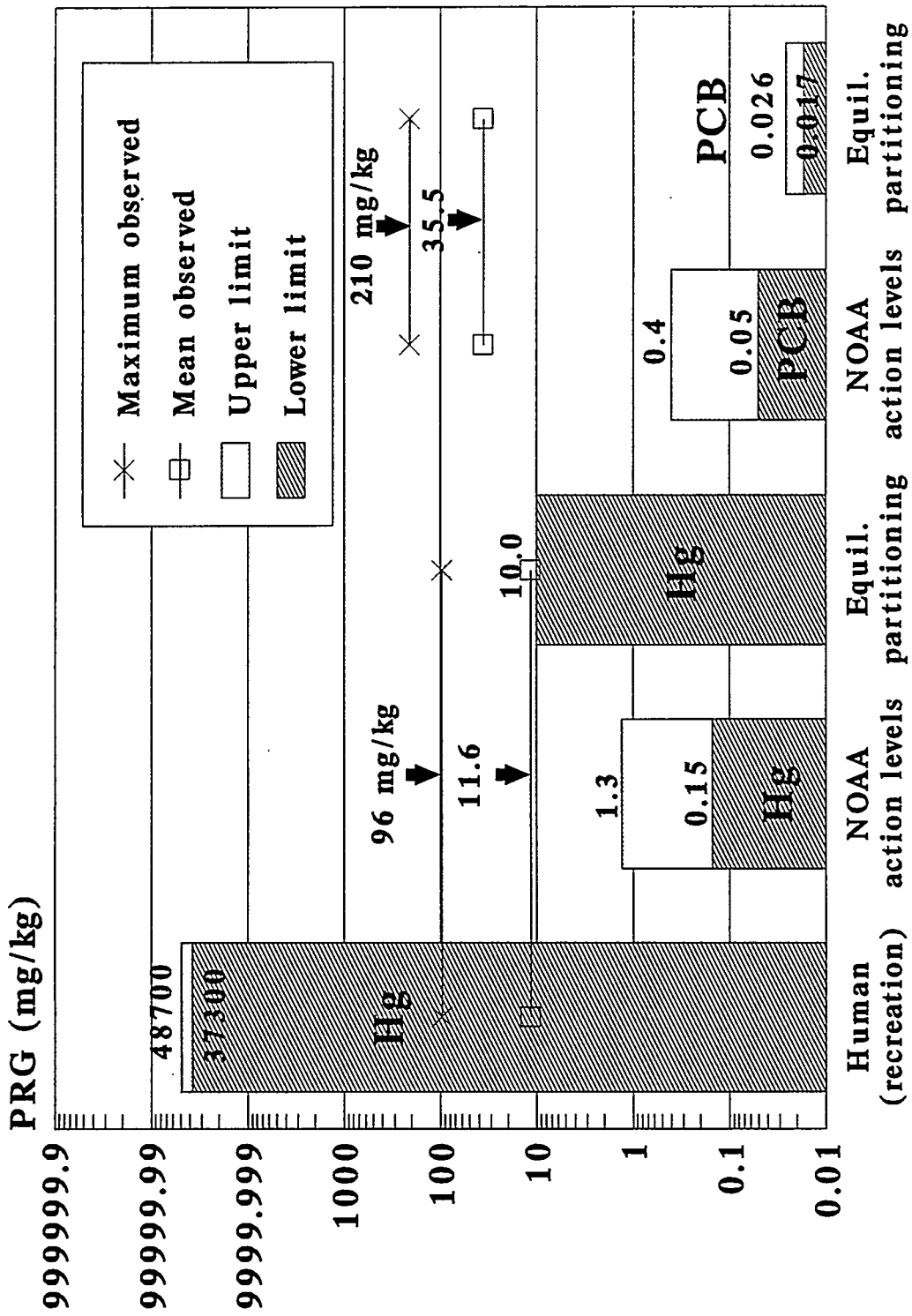

Fig. 7.5. Comparison of PRGs for mercury and PCBs in EFPC sediment.

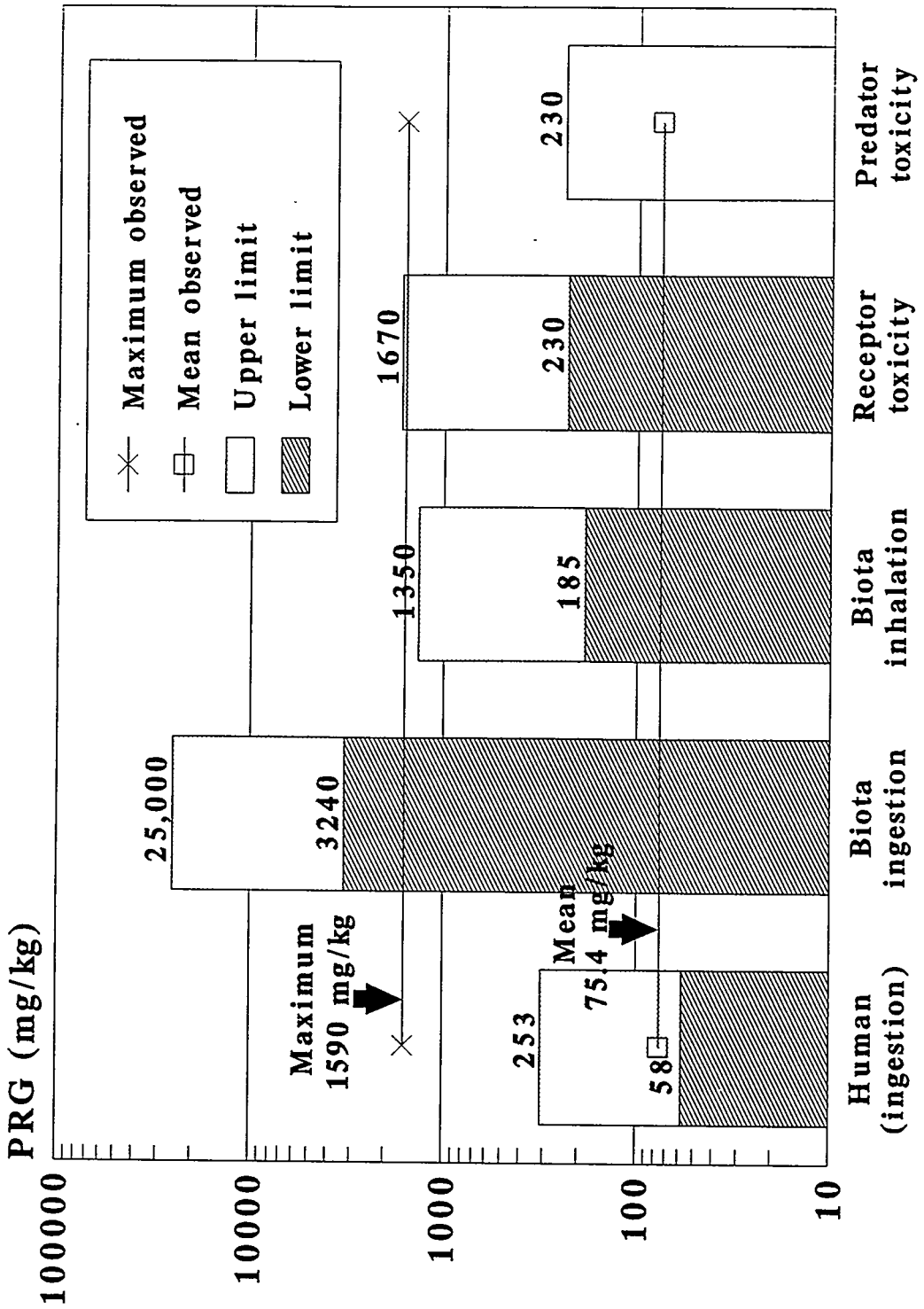

Fig. 7.6. Comparison of PRGs for mercury in EFPC floodplain soil.

7.5 SUMMARY AND CONCLUSIONS

A number of points are important in understanding and interpreting the draft PRGs that have been derived for EFPC:

- PRGs in general are derived on a chemical-by-chemical basis and do not reflect combined exposure *across contaminants*.

- PRGs for a single chemical do not reflect combined exposure *across all environmental media* in which the contaminant has been found. PRGs for a given chemical are based on a subset of all of the exposure pathways of potential concern for that contaminant. For EFPC, PRGs are derived for combined exposure across pathways that are likely to occur simultaneously. An example would be soil ingestion and dermal contact.

- Because the derivation of PRGs does not reflect combined exposure across chemicals and all exposure pathways of potential concern, PRGs cannot categorically be adopted as final remediation goals.

- Multiple PRGs may be developed for each single contaminant identified. These different PRGs reflect target goals for sets of relevant exposure pathways. In the current evaluation of EFPC, for each chemical of concern, a PRG has been derived for: soil ingestion and dermal contact using agricultural/homesteader exposure assumptions; soil ingestion and dermal contact using open land use exposure assumptions; hypothetical groundwater ingestion and inhalation exposure to volatiles; recreational use of surface water, including swimming and wading; direct contact with sediments during recreational use of the creek; ingestion of hypothetically grown produce on floodplain soils; and combined ingestion of produce, beef, and dairy exposed to contaminants in floodplain soils.

- The equations presented by EPA in RAGS Vol 1, Part B (EPA 1991c) are recommended for use in calculating risk-based PRGs at the scoping stage of the RI/FS. EPA indicates that these equations are based on standard default assumptions that may not reflect site-specific conditions. EPA specifies that when risk-based PRGs are to be calculated based on site-specific conditions, the risk assessor should modify the full equations, and/or develop additional ones. In the evaluation of EFPC, the exposure equations were designed to reflect site-specific considerations and to be consistent with the assumptions adopted in the BRA.

- The reasonable maximum exposure (RME) assumptions adopted in the baseline human health risk assessment of EFPC have been used in the derivation of draft PRGs. In

order to derive conservative estimates, most likely exposure (MLE) assumptions have not been used at this time.

- The most important considerations in deriving ecologically based PRGs are BCFs and BAFs from environmental media through the food chain to predators. There is uncertainty the accumulation factors, in the exposure amounts, and in the range and dietary mix of the predators. Exposure models for predators will have to be refined during the FS/EIS process.

It is important to recognize that the numbers provided in this section are preliminary goals and do not in any way constitute final remediation goals. Risk assessment is a dynamic, iterative process and the exposure assumptions and risk assessment methods will undergo refinement during the FS/EIS process and following review by the regulatory agencies.

8. CONCLUSIONS

Section 8 is a summary and synthesis of the results of the remedial investigation of Lower East Fork Poplar Creek (EFPC). The body of the remedial investigation report presents a detailed discussion of methods, results, and conclusions. This section encapsulates the key conclusions and recommendations of the study. Conclusions are provided in the principal areas of importance: the analysis of the nature and extent of contamination, the results of the baseline risk assessment, and the results of the baseline ecological risk assessment.

NATURE AND EXTENT OF CONTAMINATION

- The EFPC investigation began originally under Sect. 3004(v) of the Resource Conservation and Recovery Act (RCRA). The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) became the driving regulator when the Oak Ridge Reservation was placed on the National Priority List in December 1989. RCRA and the National Environmental Policy Act are integrated into the remedial investigation/feasibility study process, which requires a remedial investigation to be conducted along with a baseline risk assessment and an ecological risk assessment.

- Floodplain soils contain the highest concentrations of potential contaminants of all the environmental media (average mercury concentration of 47 mg/kg, compared to <0.12 mg/kg at the reference site). Elevated levels of inorganic trace metals, polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons, and radionuclides have been detected in the floodplain. Mercury was identified as the principal nonradiological contaminant through a concentration toxicity screening process. Uranium was found to be the principal radionuclide with elevated activity levels.

- The levels of manganese and beryllium in soils are demonstrated to be indistinguishable from background (i.e., they are clearly naturally occurring). Mercury, arsenic, and cadmium are found in floodplain soils at levels consistently above background concentrations at Hinds Creek. Mercury was confirmed as the principal contaminant of concern in EFPC floodplain soils. Arsenic and manganese are the contaminants of principal concern in groundwater.

- Because of the relatively high abundance and the lengthy release period, mercury was used as an indicator of the inorganic contaminant distribution. Distribution patterns have been mapped (with more than 3000 samples) for 5 inorganic and 2 radionuclide analytes within

the 100-year floodplain. One type of pattern is a ribbon running the length of the EFPC adjacent to (within 5-10 m of) the creek bank. The other pattern shows that contamination is not consistently distributed throughout the floodplain but occurs in well-defined contiguous areas. Four primary sites display elevated concentrations of the various inorganic analytes. Three of the areas are the National Oceanic and Atmospheric Administration, Bruners, and Sturm sites, and a fourth is in the vicinity of the Grand Cove Subdivision.

- Net volumes of mercury-contaminated soil were calculated from the contoured delineation of the contamination boundary. Volumes were estimated by layer (0-41 cm, 41-82 cm, 82-123 cm) for the $\geq$ 50 ppm and $\geq$ 200 ppm mercury classes. Only occasional points in the third layer exceed 50 ppm mercury; therefore, no volumes were calculated for this interval. As a point of reference, a total of 153,000 m^3 (5,303,000 ft^3) is contaminated in excess of 50 ppm mercury. The soil volumes decrease to 33,000 m^3 (1,171,000 ft^3) for concentrations greater than 200 ppm.

- Sediments have been used as an integrator of contamination to assess contaminant origin and the impact from watersheds adjoining EFPC. Sediments possess the same set of potential contaminants as soils, but, generally, at lower concentrations (average mercury concentration of 15 mg/kg compared to reference site = <0.12 mg/kg) and in a more heterogeneous distribution. Two classes of organic compounds, PCBs and polycyclic aromatic hydrocarbons, display bimodal spatial distributions. Higher than average concentrations of both compounds are observed at the outfall of Lake Reality and near the downstream reach of commercial development along EFPC. One tributary appears to be a source of metals, also.

- Relatively few chemical compounds in surface water were detected during base flow conditions that exceeded those at the reference site. However, mercury was observed at low (0.5 mg/kg) concentrations that decreased progressively away from Lake Reality, and isotopic uranium was observed at a low activity level. Storm flow measurements display a several-fold increase in levels of inorganic compounds due, primarily, to their attachment to suspended soil particles. No contributions of total petroleum hydrocarbons, pesticides, or herbicides have been recognized from adjoining watersheds, and the impact of the adjoining watersheds to surface water quality is considered negligible.

- Groundwater of the EFPC aquifer is contained within the unconsolidated soil horizon under water table conditions. From a survey of potential users, groundwater has not been found to be a source of potable water within the EFPC floodplain. Moreover, the use of groundwater from the soil horizon is severely limited by the impermeable nature of that horizon. No temporal or spatial gradient or plume of contamination has been observed. Groundwater has the same potential contaminants as soils, and contaminants within the groundwater are, for the most part, related to the suspended soil fraction.

- Tests performed by Oak Ridge National Laboratory researchers could not identify concentrations of mercury vapors within the EFPC floodplain in excess of ambient levels. Neither high-volume nor low-volume (but sensitive) measurements could distinguish between mercury vapor concentrations within the floodplain and at a reference site within a remote section of the Y-12 Plant reservation.

- Aerial radiation measurements were made by the U.S. Department of Energy during 1992 to determine the level of gross gamma-ray exposure calibrated to 1 m above ground surface. A review of the results showed only one small area with levels higher than background. However, there were no statistically significant differences between this area and background readings. The area correlated with a study site (the Bruners site) shown to have high concentrations of inorganic analytes. This area has been the focus of thorough sampling for radiological analyses and has not been shown to be of concern from risk-based criteria.

HUMAN HEALTH BASELINE RISK ASSESSMENT

- There is currently no imminent or substantial endangerment to human health associated with exposure to contaminants in environmental or biological media of EFPC. The results of the baseline risk assessment do not indicate the need for immediate short-term action to mitigate potential exposures or risks to human health.

- Risk assessment based on the use of the reasonable maximum exposure assumption yields very conservative results and indicates potential for adverse noncarcinogenic and carcinogenic effects associated with long-term exposure to contaminants in EFPC. Risk estimates were found to exceed the target ranges for the protection of human health established by the U.S. Environmental Protection Agency (EPA) under the Superfund program.

- For noncarcinogenic effects, risk estimates were designated of concern (i.e., exceeding the target range established by EPA) if the Hazard Quotient for any given chemical or the Hazard Index for combined exposure across chemicals exceeds 1. Estimates of excess lifetime cancer risk that exceed 1×10^{-4} were designated of concern (i.e., falling outside the target range of 10^{-6} to 10^{-4} established by EPA for waste site remediation under the Superfund Program).

- Contaminants driving the elevated risk estimates in the baseline risk assessment of EFPC include mercury, arsenic, beryllium, cadmium, and manganese. Organic chemicals observed in EFPC media did not substantially contribute to the estimated risks to human health. The risks associated with exposure to radionuclides fell within the acceptable ranges established by EPA in all cases.

- Risks exceeding EPA target ranges were associated with exposure to chemical contaminants in floodplain soils and groundwater. The exposure pathways of primary concern are: 1) inadvertent ingestion exposure to soils; 2) the food chain pathways (produce, beef, and dairy); and 3) hypothetical use of groundwater as a source of drinking water.

- Risk assessment for combined exposure across pathways for the residential and agricultural scenarios always resulted in estimates exceeding EPA target ranges. The open land use scenarios never resulted in risk estimates exceeding EPA targets. Evaluation of the commercial land use scenario was based on hypothetical use of groundwater (i.e., future use) in a business setting. Risk estimates for this pathway exceeded the EPA target range for the protection of human health.

- Three areas characterized by elevated levels of mercury in soils along the Sewer Line Beltway were included in the baseline risk assessment. An open land use scenario was the basis of the evaluation. Risk estimates for the Sewer Line Beltway were well below the target range established by EPA for the protection of human health.

- The quantitative uncertainty analysis conducted as part of the baseline risk assessment indicates substantial uncertainty surrounding the estimates of risk to human health associated with the food chain pathways. To further explore the conservatism in the food chain pathways, risk assessment was conducted using soil data from the Hinds Creek reference location and treating this location as an agricultural land use area. Even in the absence of mercury contamination, the results of the risk assessment for the background location far exceed the acceptable ranges established by EPA.

- Given the conservatism built into the exposure assumptions for the food chain pathways and the fact that agricultural activity along EFPC is very limited (and is likely to remain limited in the future), it is concluded that the reasonable maximum exposure risk estimates for the produce, beef, and dairy pathways are not a useful decision-making tool. The food chain pathways are *not* recommended as a basis for determining the need for site remediation unless additional work is conducted to derive site-specific biotransfer factors.

- Although risk estimates for the groundwater ingestion pathway exceed the acceptable range established by EPA under the Superfund program, this pathway is concluded to be of minimal concern to residents of EFPC.

  - No one is currently using groundwater in the vicinity of EFPC as a source of drinking water or is likely to do so in the future.
  - The results of the risk assessment demonstrate that unfiltered levels of inorganic chemicals are largely responsible for the elevated risk estimates exceeding the acceptable range established by EPA.
  - Exposure point concentrations determined from filtered samples did not exceed maximum contaminant levels and resulted in considerably reduced risk estimates.

  Taking into consideration the conservatism of the reasonable maximum exposure estimate and the uncertainty inherent in the exposure assumptions, the groundwater pathway should *not* be used as a basis for determining the need for site remediation.

- EPA has withdrawn the oral reference dose for mercury from the Integrated Risk Information System database. A very conservative reference dose obtained from the EPA Health Effects Assessment Summary Tables publication (FY 1992) was used in the baseline risk assessment. Alternate reference doses derived by the EFPC risk assessment team and Oak Ridge National Laboratory as part of the baseline risk assessment indicate one to two orders of magnitude additional uncertainty in the reference doses. (The site-specific reference dose for mercuric sulfide has been submitted to the EPA Environmental Criteria and Assessment Office for review). The direct implication of this finding is another one to two orders of magnitude of conservatism in the reasonable maximum exposure estimates of risks to human health.

- The results of the baseline risk assessment indicate soil ingestion as the principal exposure pathway of concern and the most appropriate basis for determining the need for site remediation. Preliminary remediation goals have been derived for contaminants in soil as well as in other environmental media at EFPC. For example, preliminary remediation goals for mercury-contaminated soil ingestion and dermal exposure in an agricultural setting are calculated to be 58 mg/kg (children) and 198 mg/kg (adults). These risk-based, chemical-specific goals are not yet final cleanup levels. They are preliminary benchmarks that assist in the initial phases of the feasibility study. Final remediation goals will be developed in the feasibility study.

ECOLOGICAL RISK ASSESSMENT

- There is substantial ongoing risk to ecological resources, particularly aquatic organisms, in the upper part of the creek from exposure to contaminants in environmental and biological media of EFPC. The results of the ecological risk assessment do not indicate a need for immediate short-term action to mitigate exposure or risks to ecological health.

- The EFPC ecological risk assessment, which is structured according to the framework for ecological risk assessment from EPA and also U.S. Department of Energy guidance, consists of four interrelated activities: (1) problem formulation, (2) exposure characterization, (3) effects characterization, and (4) risk characterization. Assessment endpoints and measurement endpoints were advanced. Approved protocols were followed to select and measure abundance and contaminant body burdens at various trophic levels in susceptible aquatic indicators (fish, benthic macroinvertebrates, and periphyton) and susceptible terrestrial indicators (small mammals, birds, annelids, insects, and vegetation).

- The ecological risk assessment uses two interrelated approaches to ecological risk characterization. In the quotient method, observations were compared to numerical criteria from various government agencies for protection of ecological resources. The second approach utilized weight-of-evidence arguments to evaluate the strength of the relationship between stressors and observed effects on indicator species and the implications for assessment endpoints. The arguments evaluate temporal association, spatial association, stressor response, strength of association, and plausibility of each proposed relationship.

- Based on a review of literature and field surveys, threatened and endangered species are not found in EFPC and the floodplain.

- Seventeen wetlands exist in the floodplain; some have mercury contamination, but there appears to be little to no negative impact on the existence of the wetlands. Other terrestrial habitats also are present and display little to no impact on their existence.

- The food chain is a primary exposure pathway for aquatic fauna. Direct contact with the surface water and sediment pore water are also exposure pathways for aquatic biota. The food chain is the most important exposure pathway for terrestrial fauna.

- The principal exposure pathways of concern for aquatic biota are ingestion of water-borne food and contact with water-borne contaminants. Releases from the Y-12 Plant are the primary source of water-borne contaminants. Additional contributions, albeit much smaller, to ecological risk come from the municipal sewage treatment plant and other point and non-point sources along the creek. Experiments on fish uptake in tanks containing mercury-laden waters from Lake Reality, both with and without a contaminated sediment substrate, establish the minor role of sediments as a source of exposure to aquatic organisms.

- The quotient method for evaluating risk to aquatic organisms showed many exceedances of the acceptable value of 1, others by a factor of more than 10, and others by a factor of more than 100. For example, criteria for contaminant body burden concentrations as advanced by the U.S. Fish and Wildlife Service and the Food and Drug Administration were exceeded by measured body burdens in EFPC fish tissue.

- Exposures to terrestrial biota come from contaminant deposits in the EFPC floodplain soils. These exposures result in substantial risk as determined by contaminant body-burden measurements and quotients in excess of 10 and 100 to predators of earthworms and insects. Predators of small mammals and birds are at risk if those predators obtain the majority of their food from the EFPC floodplain.

- All indicator organisms from EFPC, both aquatic and terrestrial, showed higher body burdens of one or more of the contaminants of concern than individuals from the reference sites. Several types of aquatic organisms showed many-fold concentrations relative to the reference site.

- Bioaccumulation was documented for mercury and other metals, PCBs, and pesticides in aquatic organisms, even when these same chemicals were below analytical detection limits in the water where the organisms were living. Concentrations of several contaminants of concern exceeded benchmark values such as ambient water quality criteria in aquatic biota, even though they were not detected in surface water.

- Methylmercury concentrations were very low in EFPC soils and sediments; however, methylmercury was assumed from Biological Monitoring and Abatement Program data and other evidence to be the predominant mercury species in aquatic biota, including flying insects. Consumption of contaminated aquatic biota provides a risk to terrestrial piscivores.

- The weight-of-evidence analysis for aquatic resources indicates that continual releases of water-borne (including dissolved and particle-bound) contaminants such as mercury, uranium, other inorganics, PCBs, polycyclic aromatic hydrocarbons, and pesticides are continuing from the Y-12 Plant and that these contaminants:

  (1) are the largest source of the elevated whole-body burdens of these contaminants in fish, benthic macroinvertebrates, and crayfish in EFPC, and especially in the upper part of the watershed;

  (2) impact fish community structure in EFPC, resulting in communities dominated by species tolerant of degraded water quality conditions; and

  (3) cause reduced taxonomic richness and diversity in the fish and benthic macroinvertebrate communities in EFPC.

  Historical data from at least three previous studies on mercury and PCB concentrations confirm this.

- Other chemical and physical stressors to aquatic organisms are or have been residual chlorine, water temperature, and stream flow (augmented, consistent flow). These types of multiple stressors are judged from weight-of-evidence analysis to be of lesser consequence under current conditions.

- Ecological risks -- both aquatic and terrestrial -- are generally (1) highest in the upper reach of the creek/floodplain below Lake Reality, (2) moderate in the middle reach, and (3) lowest in the lower reach.

- The ecological preliminary remediation goals for mercury have been estimated at 10 mg/kg for sediment and 200 mg/kg for the floodplain soils.

- Recovery of the aquatic ecological community has been occurring in the upper reaches of the creek above and below Lake Reality as documented by the Biological Monitoring and Abatement Program and the present study. Dechlorination of the process water and other remedial activities at Y-12 have lowered the concentrations of some water-borne chemical stressors; thus, exposure has decreased, followed by partial ecological recovery. Nonetheless, elevated contaminant body burdens and an excess of tolerant species are still present.

- Water-borne contamination (especially mercury and PCBs) must be controlled for protection of aquatic biota. Sediment or floodplain soil removal will not sufficiently reduce these contaminant concentrations in aquatic biota. In the feasibility study/environmental impact statement, the implications of this and other findings will be rigorously and systematically examined.